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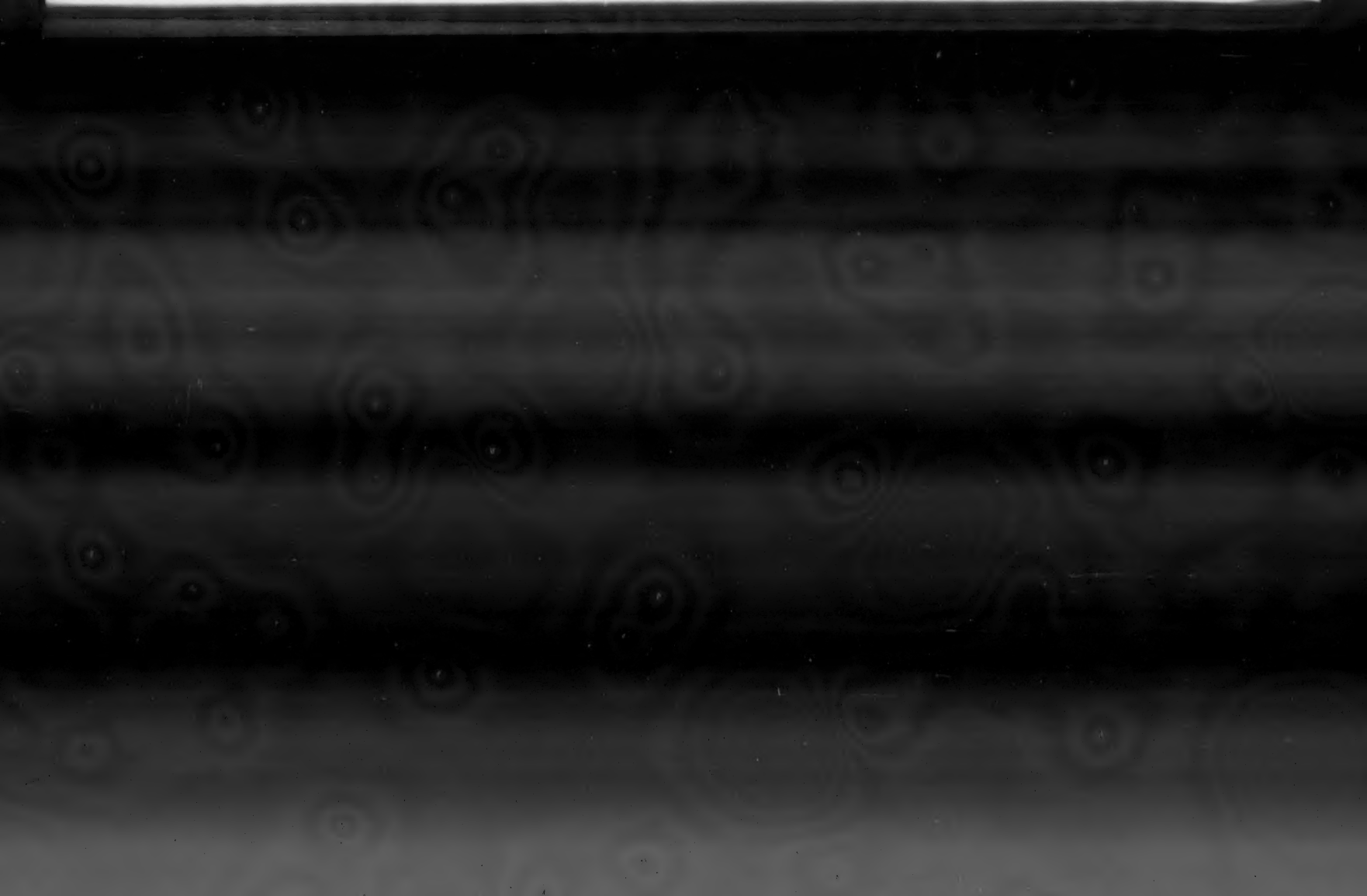

Section of Tropical Diseases and Parasitology

President—H. S. STANNUS, M.D.

[*November* 2, 1933]

Elephantiasis Nostras (Non-filarial Elephantiasis)

By Sir ALDO CASTELLANI, K.C.M.G.(Hon.), D.S.C., M.D., F.R.C.P.

DEFINITION

I USE the term "elephantiasis nostras" to indicate a condition clinically indistinguishable from filarial elephantiasis, but in which the filarial origin can be excluded with certainty. The condition, as is the case with filarial elephantiasis, is characterized by repeated attacks of pyrexial lymphangitis, followed by temporary œdema, then by solid œdema, and finally by pachydermia.

HISTORY AND GEOGRAPHICAL DISTRIBUTION

The disease has been known for many years, but, it must be admitted, somewhat imperfectly and in a somewhat confused manner, as the term "elephantiasis nostras" has been used in a very lax way to denote all kinds of elephantoid conditions of widely different ætiology, including elephantoid conditions due to climatic bubo, syphilis, tuberculosis and tumours.

The disease is found comparatively frequently in Southern Europe, but cases have been recorded from practically every continental country, and autochthonous cases occur also in England. The disease is also found in North America. In all probability it has a cosmopolitan distribution, and doubtless occurs also in the tropics and subtropics, although confused there with filarial elephantiasis.

ÆTIOLOGY

The filarial origin in my cases can be excluded with certainty, as, not only did the patients show no microfilariæ in the blood—though microfilariæ may be very rare or absent also in filarial elephantiasis—but all the cases observed by me contracted the disease in Great Britain, in Italy, in the Balkans, and in parts of the United States, where *Wuchereria bancrofti* does not exist.

In all probability the disease is of bacterial origin, as shown principally by the examination of the affected inguinal glands during the attacks of lymphangitis and

lymphadenitis. It is of no use examining the œdematous skin by deep punctures, or the glands during the *apyrexial* intervals, when, in my experience, if proper aseptic precautions are taken, no bacteria of any kind are grown. The organisms found may be separated into four groups:—

(*a*) Gram-positive cocci of the staphylococcus type (*Staphylococcus aureus, S. albus, S. viscidus*). These are probably of little ætiological importance; they are at times associated with streptococci.

(*b*) Gram-positive cocci of the streptococcus group, mostly strains of *Streptococcus hæmolyticus* type.

(*c*) Gram-negative cocci of the *Micrococcus* (coccobacillus) *metamyceticus* type.

(*d*) Cocci of the *Micrococcus myceticus* type, which are at times Gram-positive, at times Gram-negative; that is to say, the same strain regularly subcultured may at times appear to be completely Gram-positive, at other times completely Gram-negative, and most times partially Gram-positive, some cells being stained, others not stained.

These cocci of the *myceticus* type seldom, however, give rise to true elephantiasis; they generally cause an elephantoid condition characterized by the presence of slow-developing abscesses and sinuses somewhat resembling a mycosis; exceptionally they may give rise to elephantiasis.

A few words may be said on *Micrococcus metamyceticus* and *Micrococcus myceticus*, as they are little known.

These two organisms are somewhat similar; indeed, for some time I considered that the organism which I later called *metamyceticus* was merely a strain or variety of *Micrococcus myceticus*. Neither organism usually grows on plain agar when the agar is inoculated direct from the lesions, and their sugar reactions do not differ very greatly. The fact, however, that *metamyceticus* is always Gram-negative, and in certain media—especially trypsinized agar—often takes a bacillary shape, led me to separate it from *Micrococcus myceticus*.

(For further details the reader is referred to the description in the author's paper in *Arch. Derm. and Syph.*, 1928, xviii, 857.)

Pathology

The sequel of pathological changes is as follows: (1) Acute inflammation of certain lymphatics and lymphatic glands occurs, due to bacterial invasion. (2) Owing to the pathological changes produced in the lymphatics by the bacteria and their toxins, œdema develops, which later becomes solid œdema. (3) The œdematous stage is followed by hyperplasia which affects the skin and subcutaneous tissues.

The portal of entrance of the bacterium is usually an abrasion or small wound in the legs or feet, but in some cases the portal of entrance cannot be detected, and distant infective foci have been suggested, as the teeth, the tonsils or the appendix. It may be of some slight interest to note that in three cases in New Orleans the bacterial infection apparently took place through the lesions caused between the toes by epidermophytosis fungi (Mango toe, Hong Kong foot).

Morbid Anatomy

Very little is known with regard to the morbid anatomy of the disease apart from data collected at operations, but it would appear that the lesions are very similar to those found in filarial elephantiasis, the lymphatics being dilated and the inguinal glands of the affected side being often enlarged, and there may be œdematous peri-adenitis. In cutting into the skin of the affected limb it will be noted that the skin is greatly thickened, and in the subcutaneous tissue there will be found dense

fibrous trabeculæ, with the spaces filled with a yellow, oily, blubbery, fatty substance which exudes lymph.

Histopathology

Epidermis.—With regard to the epidermis, the stratum corneum is hardly thickened in cases of elephantiasis nostras glabra, while it is thickened in cases of elephantiasis nostras verrucosa. The rete Malpighii layer is not usually thinned in elephantiasis nostras glabra, but it is very greatly thinned in elephantiasis verrucosa. In both types the nuclei of the Malpighian layer are usually very close together, this showing great diminution or absence of cytoplasm in the cells; the "prickles" in many cases are not seen. In the verrucose type, finger-like out-growing processes are noted, covered by a loose thickened horny layer.

Corium and subcutaneous tissue.—These are greatly thickened, and here and there little accumulations of small round cells are at times seen, especially in the papillary layer. The open lymph spaces are usually very prominent and very numerous; the arterioles are greatly thickened, the intima showing special hypertrophy; there is seldom thrombosis. The connective tissue in most cases is very loose and looks œdematous; in parts the cells are branched as seen in mucoid degeneration.

In brief, the principal histological features of the skin in elephantiasis nostras are: (1) Close approximation of the nuclei in the Malpighian layer, and great diminution or absence of cytoplasm with almost total absence of the "prickles." (2) Large number of prominent open lymph spaces. (3) Thickened arterioles. (4) Fibrosis.

Lymphatic glands.—If a gland is removed during or soon after the attack of lymphangitis, it will be found enlarged and somewhat congested. The capsule is slightly thickened, and the trabecular septa seem to be slightly hypertrophic. In the substance of the gland, numerous small vessels may be seen, with thickened walls and occasionally thrombosis. The lymphatic tissue proper does not usually show much alteration. In chronic cases there is much fibrosis and the lymphatic tissue may be atrophied.

Symptomatology

The disease begins with an attack of lymphangitis, usually of the leg, with high fever; the patient may feel very ill and there may be great prostration. On the affected limb, red erysipelas-like patches are seen, which, however, are seldom marginated as is the case with true erysipelas, the edges being rather indefinite. The condition may extend up or down the limb. In some cases the redness is linear or in streaks. The affected limb becomes œdematous and there may be pain and tenderness. The regional glands are generally enlarged and painful. Although after the first attack of lymphangitis the swelling may disappear and the limb may resume its normal size, this does not happen after repeated successive attacks, and gradually the limb becomes more and more swollen, while the natural folds, especially the ankle fold, become much exaggerated by the swelling on either side, so that deep sulci are formed. The dorsum of the foot becomes swollen and puffy and is separated by the deep ankle sulcus from the swollen lower part of the leg.

At first the skin is smooth and soft, forming the smooth variety of the complaint "elephantiasis nostras glabra," which may in some cases persist indefinitely or may later become hard, thick and rough, being elevated into bosses or warty elevations, often of a dark colour, forming the verrucose variety, "elephantiasis nostras verrucosa." The appendages now atrophy from malnutrition, the hairs may drop off, and the nails become rough and thickened, while the skin perspires less and the sensibility is diminished. The swelling is more commonly met with below the knee, but the whole thigh may become implicated.

If left untreated, the size of the leg gradually increases with repeated attacks of fever, and may reach enormous dimensions. After some years the attacks of fever may cease altogether, though the condition of the leg usually remains unchanged.

Regions of the Body Affected

These seem to be the same as in filarial elephantiasis, the most common situation being the lower limbs. The scrotum is affected fairly frequently, the upper limbs very seldom, the face and lips occasionally. When the disease affects the scrotum it begins usually with recurring erysipelatous attacks, with a red inflammatory blush on the skin, and fever. After each attack the scrotum is larger than before and it goes on increasing until it reaches an enormous size. The penis itself is often buried in the tumour. The testicles are situated at the upper and back part of the tumour and may be surrounded by hydroceles.

On examination by X-rays, the bones of the affected leg do not seem, in the majority of cases, to be hypertrophied; in others a slight hypertrophy may be noted.

Course

This is chronic and progressive and there is no tendency to spontaneous cure.

Diagnosis and Prognosis

As already stated, it is impossible to differentiate clinically elephantiasis nostras from filarial elephantiasis, as in both conditions there are repeated attacks of lymphangitis followed by temporory œdema, then solid œdema and finally pachydermia with great hypertrophy of the skin and subcutaneous tissues.

In elephantiasis nostras the blood never shows the presence of microfilariæ, but microfilariæ are absent from the blood in many cases of filarial elephantiasis also. In elephantiasis nostras there is no eosinophilia, but eosinophilia is far from being a constant feature of filarial elephantiasis and is practically always absent in the pachydermic stage.

In practice the diagnosis of elephantiasis nostras is based chiefly on the knowledge that the patient contracted the disease in a country where filaria is absent, and also on the negative results of the serological and skin tests for filariasis.

The prognosis is unfavourable, although life is seldom endangered.

Treatment.

In the *first stage* when repeated attacks of lymphangitis occur, salicylates given internally are at times useful: 10 to 15 grains of sodium salicylate, with or without the same amount of sodium bicarbonate in a large amount of water or milk three times a day, or salicin gr. 10 three times a day, may be administered. The salicylates may be given also by intravenous injection.

During the *afebrile periods*, autogenous vaccines are of use, but should be given in small doses. In cases in which *Micrococcus metamyceticus* is found, vaccines prepared with this coccus seem to be especially useful and generally stop further attacks of fever, but naturally the elephantoid condition due to fibrotic processes does not respond to the vaccine and does not disappear. The vaccine should be given every fourth or fifth day in small doses—10 to 100 millions.

Protein-shock treatment has been tried in some cases with very little or no benefit in my experience.

In the *pachydermic stage*, when attacks of fever no longer occur, I advise giving once or twice a year a period of treatment lasting three or four weeks, consisting of complete rest in bed, bandaging and injections of fibrolysin, as is done in filarial elephantiasis in the same stage. It is a mistake to give fibrolysin during the acute stages.

Surgical treatment.—This is the same as for filarial elephantiasis. It is not very satisfactory except when the condition is localized to the scrotum, when an operation may be performed successfully, as in elephantiasis of the scrotum of filarial origin. Any operative intervention should be preceded by a course of vaccine.

Discussion.—Dr. P. H. MANSON-BAHR said that the subject of elephantiasis must be studied as a whole, because although it was agreed that the causes of lymphatic blockage were in several instances fundamentally different, yet their end-results were essentially the same. He had formulated in his own mind a rough kind of classification to enable him to understand the subject. He considered that the causes of elephantiasis of the arms or legs could be stated as follows:—

(*a*) Congenital or familial elephantiasis. (Milroy or Meige's Disease. Congenital stenosis of the main lymphatic trunks.)

(*b*) Parasitic elephantiasis, *Filaria bancrofti* or *Onchocerca volvulus*.

(*c*) Septic elephantiasis. (Elephantiasis nostras, lymphatic infection by streptococci or other organisms.)

(*d*) Toxic elephantiasis, due to chronic absorption of irritating toxins, such as chrysarobin.

(*e*) Obstructive elephantiasis, due to carcinomatous growths, syphilitic ulceration or yaws.

(*f*) Venous elephantiasis, due to thrombosis of venous trunks such as *Phlegmasia alba dolens* in women, and certain cases of elephantiasis of arms and legs described in Germany and secondary to chronic venous obstruction.

He considered it at present very necessary to ascertain whether attacks of lymphangitis were due to sepsis or were merely allergic phenomena, as they well might be, in filarial disease.

Dr. H. MACCORMAC: It is held by many that the factors necessary to complete the formula for the development of elephantiasis are an inflammatory reaction and an interference with the venous and lymphatic return. The inflammatory phase is well known to dermatologists in the so-called recurrent erysipelatoid eruptions of the face which often resulted in solid œdema of the eyelids and occasionally elsewhere. This inflammatory reaction is not observed, or not observed to the same degree, in the cancerous pachydermia of the arm. Handley's original view that this deformity depended on lymphatic permeation has undergone modification, and he now states that it has been shown by Starling and others that obstruction of the lymphatics alone does not cause œdema. He further says that the inflammatory reaction and subsequent fibrosis which obliterated the lymphatics are likely to press upon and obstruct the concomitant veins.

In the case of elephantiasis affecting the legs in syphilitics a similar lymphatic and venous obstruction is present, causing the characteristic deformity which is occasionally observed in a dermatological department.

Dr. F. PARKES WEBER alluded to the differential diagnosis of the congenital-developmental group of chronic œdema of limbs, described first by Nonne of Hamburg, and shortly afterwards by Milroy in America, and somewhat later by Meige and others in France. He said that the diagnosis might be difficult owing to the fact that there was often no history of similar cases in the family and that even in familial cases the œdema did not always show itself at birth, or even during infancy. Moreover, cases of this group sometimes also came to be complicated with recurrent erysipelatoid attacks.

Dr. HAMILTON FAIRLEY said that in filarial elephantiasis the lymphangitis might be associated with focal nodes as stressed by O'Connor; on section these were found to consist of masses of dead filarial worms. Secondly, the intradermal test with dirofilarial antigen, if positive, favoured a filarial origin.

Dr. R. T. BRAIN, by courtesy of Dr. W. J. O'Donovan, gave details of a case of elephantiasis nostras which illustrated the difficulty of the ætiological problem. There was no history in this case of lymphangitis or of fever.

Dr. W. E. COOKE said he was interested to hear that autogenous vaccines had been found useful in certain of these patients. In one particular case he had given four injections of T.A.B. intravenously, thereby producing protein shock. The improvement in this case was permanent, as the patient had been seen five years later.

Dr. H. CORSI said he considered elephantiasis to be the end-result of any process which obliterated the fine distal lymphatics.

Section of the History of Medicine

President—Sir StClair Thomson, M.D.

[*December* 6, 1933]

On Infectious Diseases and Epidemiology in the Hippocratic Collection

By E. W. Goodall, O.B.E., M.D.

I propose in the following pages: (1) To discuss what infectious diseases are mentioned in the Collection, and (2) to state the views which its writers held on the causes of epidemics. The quotations are from the English translations of the works of the Collection in the *Loeb Library* and in Adams's edition. For the works not included in these I refer to Littré's French translation.

Although several diseases which are now known to be infectious are distinctly described in the Collection, yet nowhere in these works is infection alluded to, much less explained. Why that should be is not known. It cannot be due to ignorance on the part of the writers, for the notion of infection or contagion was entertained amongst certain of the peoples of the Near East not only during but even before the period in which the Hippocratic writings were penned. It may be, however, that the authors of the Collection were anxious to avoid even a suspicion of superstition in what they taught.

That some of the diseases mentioned or described in the Collection attacked more than one person at the same time and in the same locality is clear. While it is true that the simultaneous affection of many persons by the same disease is not necessarily a proof that the disease is infectious, yet we have no reason to suppose that such epidemic diseases as we now know to be of microbic origin were not also of the same origin when they occurred in the past.

Of the infectious diseases mentioned in the Collection there are three which can be identified with certainty, namely, *mumps*, *puerperal fever* and *erysipelas*.

Mumps.—There was an epidemic in Thasos. "Many had swellings beside one ear, or both ears, in most cases unattended with fever, so that confinement to bed was unnecessary. In some cases there was slight heat, but in all the swellings subsided without causing harm; in no case was there suppuration such as attends swellings of other origin. This was the character of them: flabby, big, spreading with neither inflammation nor pain; in every case they disappeared without a sign.[1] The sufferers were youths, young men, and men in their prime, usually those who frequented the wrestling school and gymnasia. Few women were attacked. Many had dry coughs which brought up nothing when they coughed, but their voices were hoarse. Soon after, though in some cases after some time, painful inflammations occurred either in one testicle or both, sometimes accompanied with fever, in other cases not. Usually they caused much suffering." (*Epid.* I, i.)

It is worthy of note that as in recent times so in the days of Hippocrates, information concerning mumps was obtained mostly from outbreaks amongst assemblies of males (soldiers, schoolboys, especially the former).

Puerperal fever.—Of the forty-two cases of which details are given in *Epid.* I and III, eight were undoubtedly cases of *puerperal fever*.[2] These all followed delivery or miscarriage and all except one were fatal.

1 "That is, with no symptoms indicative of a crisis." (W. H. S. Jones.) The term "crisis" in the works of the Hippocratic era had a much wider meaning than it has at the present time; it was even applied to the termination of an illness, however it was brought about. See the definition in the work *On Affections*: (Littré, tom. 6, 216).

2 *Epid.* I, cases iv, v, ix; *Epid.* III, first series, cases x, xi, xii; second series, cases ii, xiv.

Erysipelas.—In *Epid.* III, iv, is an account of this affection, which is described as starting from small wounds and spreading all over the body. It may result in suppuration and gangrene, which sometimes involve the loss of much tissue and even of a limb. Included in this description are cases of phlegmonous inflammation, which we now know to be of the same bacterial origin as erysipelas. Case ix in *Epid.* I, is without doubt one of erysipelas: "Crito, in Thasos, while walking about, was seized with a violent pain in the great toe. He took to bed the same day with shivering and nausea; regained a little warmth; at night delirious. Second day: Swelling of the whole foot, which was rather red about the ankle, and distended; black blisters; acute fever; mad delirium. Alvine discharges unmixed, bilious and rather frequent. He died on the second day from the commencement."

In *Prognostic* xxiii will be found a short description of what is probably erysipelas of the fauces and neck, but with this I will deal later.

I now turn to certain diseases concerning which less certainty has been expressed. Undoubtedly malaria in various forms and phthisis are described in the Collection, but as these diseases are not at the present time usually classed amongst the acute, infectious diseases, I shall not treat of them. The question is, therefore, which, if any, of the acute, infectious fevers, did the Hippocratic writers meet with.

In *Epid.* I, xxi, there is a very brief discussion on crises. In it will be found an account of the illnesses of two brothers. They "fell sick together at the same time, and lay ill near the bungalow of Epigenes. The elder of these had a crisis on the sixth day, the younger on the seventh. Both suffered a relapse together at the same time with an intermission of five days. After the relapse both had a complete crisis together on the seventeenth day." The writer then goes on to say that the great majority of patients "had a crisis on the sixth day, with an intermission of six days followed by a crisis on the fifth day of the relapse," and to state the variations in the length of the interval and in the day of incidence of the crisis. The disease here described can be hardly any other than *relapsing fever*, of which, since there is reference to *a majority* of the patients, there was evidently an epidemic.

It may be objected, first, that these were cases of malaria which without question was very prevalent in the Near East in the days of Hippocrates. Now the writer of *Epid.* I and III refers to febrile affections which appear to have been malarial (see *Epid.* I, ii, v, vi, xxiv, and *Epid.* III, xii). These fevers are mentioned under the names quotidian, tertian, semitertian, quartan and so on. But in the passages under discussion (*Epid.* I, xx and xxi) there is no mention of these names, so that it may be concluded that the fevers therein referred to were recognized by the writer as being different from those described as being quotidian, tertian and the like.

In the second place, it may be objected that as *Epid.* I, xx, begins with the words "The painful swellings by the ears in fevers in some cases neither subsided nor suppurated when the fever ceased with a crisis," these supposed cases of relapsing fever could not have been of that nature, because parotitis is far from being always present in that disease. To this I reply that the writer is discussing the nature of crises rather than that of parotitis in fevers, and that it may be concluded from the following passage in the next section (xxi), that not all the patients suffered from parotitis: "Those who had the swellings by the ears had a crisis on the twentieth day, but these subsided in all cases without suppuration, being diverted to the bladder." Parotitis is an occasional complication of relapsing fever. My view is that Hippocrates saw, amongst the diseases occurring during the period he was referring to, not only relapsing fever, but also enteric, and possibly also mumps, and that he did not in his account differentiate more particularly between them, because they were all, to him, part and parcel of the results of one "constitution."

The learned translator of most of the Hippocratic works comprised in the *Loeb Classical Library*, Mr. W. H. S. Jones, states in his general introduction (vol. i, p. lvi),

that "it is extremely doubtful whether typhoid was present in Greece" at the time when these works were written.[3] He is evidently of the opinion that the bulk of the febrile diseases consisted of malaria in its various forms. He considers himself to be strengthened in his view as to the absence of typhoid by a passage in pseudo-Aristotle (*Problems*, VII, 8), as follows: "Why are those who come near the sick attacked by phthisis and ophthalmia and itch [psōra], and not by dropsy and fevers and apoplexy or the other diseases?" Fevers, that is to say, are not infectious; therefore, as typhoid is infectious, it could not have been one of the fevers alluded to by pseudo-Aristotle, and so was not present in Greece and its neighbourhood three to five hundred years B.C. I may point out, however, that up to a century ago it was strongly denied by all French and German physicians that continued fever was infectious (or contagious), while British physicians contended that it was. The reason for this marked difference of opinion was that under the term "continued fever" were included typhoid and typhus. Typhus, which was recognized to be infectious, was very prevalent in the British Isles, while it was rare in France and Germany. Even when the two diseases were differentiated clinically rather less than a century ago, both British and Continental physicians required much persuasion before they would admit the infectiousness of typhoid also. All that can be concluded from pseudo-Aristotle's statement is that he met with little or no typhus. Typhoid is not so obviously an infectious disease as typhus, smallpox and measles, the two last of which are not even hinted at in the Hippocratic Collection.

There are several places in the *Epidemics* in which fevers are described in such terms as lead me to suppose that they can hardly be any other than *enteric*. There are descriptions of outbreaks, and there are records of individual cases. I will deal first with the former, especially because in the three passages in which they are described Hippocrates definitely separates a form of continued fever from other diseases.

First, in *Epid.* I, iii, occurs the following passage: "But when summer came, and during autumn occurred many continuous but not violent fevers, which attacked persons who were long ailing without suffering distress in any other particular manner; for the bowels were in most cases quite easy, and hurt in no appreciable extent. Urine in most cases of good colour and clear, but thin, and after a time, near the crisis, it grew concocted. Coughing was slight and caused no distress. No lack of appetite; in fact it was quite possible even to give food. In general the patients did not sicken, as did the consumptives, with shivering fevers, but with slight sweats, the paroxyms being variable and irregular. The earliest crisis was about the twentieth day; in most cases the crisis was about the fortieth day, though in many it was about the eightieth. In some cases the illness did not end in this way, but in an irregular manner without a crisis. In the majority of these cases the fevers relapsed after a brief interval, and after the relapse a crisis occurred at the end of the same periods as before. The disease in many of these instances was so protracted that it even lasted during the winter.[4]

Now it is to be observed that these fevers occurred during the first "constitution" of the weather prevalent in Thasos over a period of about one year. In the spring there had been the famous epidemic of mumps. From early in the summer and on to the winter there were many cases of consumption, frequently fatal. The "con-

[3] In this he follows Littré, who, in his commentary on *Epid.* I and III, came to the conclusion that the fevers described therein differed from the continued fevers of France (tom. 2, 566). Littré wrote further: "J'ai dans l'article *dothiénentérie du Dictionnaire de Médecine*, deuxième edition, t. 10, essayé un rapprochement de quelques observations des *Epidémies* avec notre fièvre typhoïde, à tort, comme on le voit." (*Ibid.* t. 2, 157, footnote).

[4] The Greek of the word translated *continuous* is *sunechěs*. It is interesting that this word persisted in the forms "synocha" and "synochus," as a designation of "continued" fever, a fever which was not phthisical or malarial, right down to the middle of the nineteenth century. But Hippocrates uses the word "continuous" of the fever of consumption in *Epid.* III, xiii, and of malarial fever in *Epid.* I, vii.

tinuous but not violent fevers" had been prevalent also during the summer and autumn, and even to the winter, apparently at the same time as the consumptions. In chap. ii, the writer particularly distinguishes these fevers from consumptions, for he alludes to them as "the other complaints which will be described presently." From the description given it is evident that these fevers were not malarial. The shortest duration of a case was twenty days, the longest, eighty. Typhus, therefore, may be excluded. As "the bowels were in most cases quite easy," dysentery may also be set aside. So that only *enteric* (typhoid and paratyphoid), and *undulant fever* are left.

The second passage will be found in the account of the second "constitution" in *Epid.* I, v. After mentioning the symptoms of certain cases of consumption the writer says: "When autumn came, and during winter, continuous fevers—in some few cases ardent—day fevers, night fevers, semitertians, exact tertians, quartans, irregular fevers. Each of the fevers mentioned found many victims." In chap. vii the symptoms of these continuous fevers are described. They "were altogether continuous and never intermitted at all, but in all cases grew worse after the manner of semitertians, with remission one day followed by exacerbation during the next. . . . beginning mildly, and on the whole increasing always, with exacerbation, and growing worse, they had slight remissions followed quickly after an abatement by more violent exacerbations, generally becoming worse on the critical days. All patients had irregular rigors that followed no fixed law, most rarely and least in the semitertians [? semitertian-like fevers],[5] but more violent in the other fevers." The chief symptoms were copious sweats, chill in the extremities, sleeplessness followed by coma, bowel disorder, and cough. Most of the cases were protracted and had no crises. Some had relapses. There was great variety in the symptoms. In most cases there was complete loss of appetite. Wasting was also frequent.

The third passage is in *Epid.* I, xxiv and xxv. "Some fevers are continuous, some have an access during the day and an intermission during the night, or an access during the night and an intermission during the day; there are semitertians, tertians, quartans, quintans, septans, nonans. The most acute diseases, the most severe, difficult and fatal, belong to the continuous fevers. The least fatal and least difficult of all, but the longest of all, is the quartan." The writer is here referring to another, the third, seasonal "constitution." He then writes in more detail about the various malarial fevers, and in chap. xxv goes on as follows:—"Each of these fevers has its modes, its constitutions [katastasiĕs], and its exacerbations. For example, a continuous fever in some cases from the beginning is high and at its worst, leading up to the most severe stage, but about and at the crisis it moderates. In other cases it begins gently and in a suppressed manner, but rises and is exacerbated each day, bursting out violently near the crisis. In some cases it begins mildly but increases and is exacerbated, reaching its height after a time; then it declines again until the crisis or near the crisis. These characteristics may show themselves in any fever and in any disease." This is an excellent description of the first stage of an attack of enteric fever.

Amongst the forty-two cases recorded in *Epid.* I and III there are several which I find it difficult to believe were any other than enteric fever, namely, *Epid.* I, case x; *Epid.* III, case ii (first series); cases i, vii, ix, and xiii (second series). It is to be remembered that in none of the acute infectious diseases are complications more varied and numerous than in enteric fever, and that mixed infections may occur, such

[5] On this word the translator, Jones, has the following note:—"I take the pronoun οὗτος [hŏutŏs, this] throughout this chapter to refer to the remittent semitertian, or to sufferers from it." With all due deference I suggest that it refers not to an actual semitertian but to that form of continuous fever which "grew worse after the manner of semitertians." I would therefore translate hŏutŏs by the words "semitertian-like fever." It may be worth mentioning that Adrian Spigelius, who in 1645 was the first to describe the *post-mortem* appearances of typhoid fever, does so in a book entitled *De Febre Semitertiana*.

as enteric and dysentery, enteric and malaria. Deafness, which is specially mentioned in three of the cases just referred to, is not at all uncommon in severe typhoid. It may be absolute, but passes off as the patient recovers.

Other accounts of what read suspiciously like enteric will be found in *Epid.* IV, pars. 23, 25 and 55, and in *Epid.* VII, pars. 1, 3, 10, 11 and 43 (Littré's edition). *Epid.* VII, par. 1 refers to prevalent fevers of eleven to twenty-two days' duration. In pars. 3, 11, and 43 the cases are related in some detail. Par. 10 gives the case of one Chartades. This man had fever (*kausŏs*), bilious vomiting, diarrhœa, insomnia and enlarged spleen. On the ninth day he passed in two consecutive stools over five litres of blood, liquid and in clots, after which he sank into the condition commonly met with after severe hæmorrhage in typhoid and died on the same day.

A consideration of all these cases together leads me to the definite conclusion that the narrators of them were dealing with enteric. I quite realize the fact that in some of the cases, even in that of Chartades, the symptoms may be ascribed to malaria.

While perusing these narratives two points should be remembered. The first is that the Hippocratic writers referred diseases to certain weather conditions which they called "constitutions." If, therefore, epidemics of two or more diseases are concurrent, as sometimes happens, the various symptoms are ascribed to the prevalent "constitution," and the two or more epidemics are regarded as one single epidemic proper to that "constitution." It is really remarkable that Hippocrates does draw the distinctions between diseases that I have referred to above. That he did so is a proof that diseases with sufficiently obvious differences existed during one and the same "constitution."

Secondly, enteric fever varies very much in its severity, symptoms and complications from year to year.

Typhus.—In the treatise *On Internal Diseases* no fewer than five fevers are described under the name "typhus." One of them seems to have been acute rheumatism, and two may have been typhus or enteric. These two fevers are described as often coming to a crisis, sometimes suddenly, on the seventh or fourteenth day, but they might persist till the twenty-fourth or longer. In several other places in the Collection mention is made of a fever which reaches a crisis on the fourteenth day. In *Prognostic* xiv, the author, writing of a fever which is not mortal states that the sufferers from it "begin to feel pain on the fifth day, and the symptoms proceed after the manner I have described, the disease reaches a crisis on the fourteenth day." *Aphorism* 23 of Section II runs as follows:—"Acute diseases come to a crisis in fourteen days." This suggests, but, I admit, does not necessarily mean that they come to an end in fourteen days. The most important reference, however, occurs in *Epid.* II, iii, par. 1, in which a description is given of a fever which was prevalent during one summer at Perinthos, a town on the north shore of the Sea of Marmora. This fever came to an end usually about the fourteenth day, in a few cases with a sweat, in a few with shivering. In some cases an eruption appeared on the seventh, eighth or ninth day consisting of miliary asperities *like* gnat-bites. These were evidently not actual gnat-bites. This rash, however, was seen only in women. Other symptoms were diarrhœa, sweating, deafness, coma, and insomnia. The spleen was not enlarged. The disease was not fatal. Relapses were uncommon.

The narrative suggests an outbreak of typhus of a mild sort, not exactly, I admit, like the typhus in Europe during the last century. But infectious diseases vary very much in their severity and symptoms from time to time, as we ourselves have seen of recent years in the cases of scarlet fever, smallpox and even, in some parts of the world, of typhus. It is curious that in the Perinthos epidemic the eruption was confined to the female sex. However, it is related in *Epid.* VII, par. 83, that Pherecydes, who died on the ninth day of a somewhat similar fever, had on the eighth day a rash resembling gnat-bites.

In *Epid.* IV, par. 14, is recorded the cases of the wife of Nicŏstratŏs, who suffered from an acute fever of fourteen days' duration.

That *dysentery* was known to the writers of the *Collection* is in my view fairly certain, for in *Epid.* I, v, and xv, and in *Epid.* III, viii, a disease is described in which there was a severe, painful diarrhœa, with tenesmus, colic and frequent evacuations which were sometimes bloody, sometimes thin and watery.

It is stated in the *Collection* that some of the continued fevers were of very long duration, upwards of eighty and even a hundred days. Amongst them may well have been cases of *undulant fever.* The 44th of the Fourth Section of the *Aphorisms* reads as follows : " Sufferers from protracted fevers are attacked by tumours or by pains at the joints." An aphorism is a general statement based on many observations and long experience, so that the author of this aphorism and the one quoted previously as suggestive of typhus, must have met with a number of cases of continued fever, in one group of which swelling of or pains in the joints, and in another a crisis on or about the fourteenth day, struck him as a prominent feature. It must be admitted, however, that the fever with pains in the joints may have been subacute, but prolonged, rheumatic fever.

From *Prognostic* xxiii, it is clear that Hippocrates met with cases of acute and serious inflammation of the fauces and neck in which the larynx might also be involved, cases which we should recognize as being of an infectious, though not of a highly infectious, nature. The relevant portion of the section is as follows: " An ulcerated throat with fever is serious, but if some other symptom also supervene that has already been classed as bad, forecast that the patient is in danger. Angina is very serious and rapidly fatal, when no lesion is to be seen in either throat or neck, and, moreover, it causes very great pain and orthopnœa ; it may suffocate the patient even on the first day, or on the second, third or fourth. Such cases as show swelling and redness in the throat, while they are generally similar, and cause pain, are very deadly, though they tend to be more protracted than the former. When throat and neck are both red, the illness is more protracted, and recovery is most likely should neck and chest be red and the erysipelas does not turn back inwards. Should, however, the erysipelas disappear neither on the critical days nor with the formation of an abscess on the exterior, and if the patient should not cough up pus easily and without pain, it is a sign of death or of relapse of the redness. The most hopeful sign is for the redness to be determined as much as possible outwards; but if it be determined to the lungs it produces delirium, and such cases usually result in empyema." Case vii (first series), of *Epid.* III, may also be given in this connexion : " The woman suffering from angina who lay sick in the house of Aristion began her complaint with indistinctness of speech. Tongue red, and grew parched. *First day :* rigor, acute fever; shivered and grew hot. *Third day :* rigor; acute fever; a reddish hard swelling in the neck, extending to the breast on either side ; extremities cold and livid, breathing elevated ; drink returned through the nostrils—she could not swallow—stools and urine ceased. *Fourth day :* general exacerbation. *Fifth day* : death." This case appears to be more like Ludwig's angina than any other disease.

The accounts of these diseases are not related in sufficient detail for us to make a certain diagnosis. It is clear, however, that the narrator saw cases not only of the simpler and less dangerous ulcerations of the fauces, such as tonsillar ulceration and Vincent's angina, but also of the more serious forms which are nowadays comprised under the term " acute, septic inflammation of the fauces," cellulitis, erysipelas and Ludwig's angina. He possibly also met with scarlet fever with cellulitis of the neck and secondary involvement of the larynx. The condition described in the sentences beginning " Angina is very serious " may be the low-seated peripharyngitis with invasion of the larynx or œdema of that organ to which Sir James Dundas-Grant has drawn attention,[6] or diphtheria showing itself first in

[6] *Proc. Roy. Med. Chi. Soc.*, 1895, vii, 150, and *Lancet*, 1932 (ii), 158.

the larynx (*croup d'emblée*). In respect of the reference in this passage to erysipelas, *Aphorism* 25 of Section VI may be quoted: "When erysipelas that spreads externally turns inwards it is not a good thing, but it is good when internal erysipelas turns outwards."

There are, however, as has been pointed out by Haeser[7] and Souques,[8] references to what seems to have been *diphtheria* in two other works of the Hippocratic Collection. The first is in the little treatise *On Dentition*. The last two paragraphs refer to cases in which ulceration of the tonsils spreads to adjacent parts. In such cases an alteration in the character of the voice or dyspnœa occur. The cases in which there is dyspnœa are the worst.

The other passage, and the strongest in favour of diphtheria, will be found in *Epid.* II, Sect. ii, par. 24 (Littré, tom. 5, pp. 94 and 95), in which the writer describes certain sequels of cynanche. Amongst these are difficulty in swallowing, escape of fluids through the nose when drinking, and a nasal voice. Some patients suffered from one-sided paralysis of the face, mouth and palate, and lower jaw. In other cases there was inability to stand upright. These observations very strongly suggest that diphtheria with its subsequent paralysis was known to the writer of *Epid.* II.

In *Epid.* VI, vii, par. 1 and 10, an account is given of an epidemic which occurred one winter in Perinthos. The chief phenomena were cough, peripneumonia and relapses. Some of the cases were complicated with paraplegia, nyctalopia, angina, otitis and other affections. This sounds very like *influenza*.

In *Epid.* I, xiv, mention is made of an epidemic of paraplegia. Some of the cases were rapidly fatal. None of the patients suffered from any other disease.

A few of the cases in the influenza epidemic at Perinthos referred to above were complicated with paraplegia. Thus a woman had paralysis of the right arm and left leg (*Epid.* II, ii, par. 8). In *Epid.* IV, par. 50, we are told that those who, suffering from the cough, fatigued their arms, got paralysis of the arm, while those who rode or walked got paralysis of the hip and thigh. Two boys had paralysis of the right arm. By *paraplegia* is meant not only what we call paraplegia, but also hemiplegia and monoplegia. The epidemic and cases of paraplegia suggest *acute poliomyelitis*.[9]

In *Aphorism* 55 of Section IV and in *Epid.* II, iii, par. 5, fevers with abscesses in the groin or with buboes are referred to as being evil. Possibly allusion is made to *bubonic plague*.

Epidemiology.—Before proceeding to relate what is said on this subject in the Collection, I think it desirable to clear away a false notion which has been in existence for centuries, and is to be found in more than one recently written book, namely that Hippocrates postulated the existence of an unknown agent which he termed "something divine," (ti thĕiŏn), to account for the origin of epidemics, and for any peculiarity in the nature of diseases.

The origin of this notion is to be found in the book *Prognostic* of Hippocrates. I quote Adams's translation because, for the reason given below, Jones omits one sentence of the passage. It runs as follows: "but since men die, some even before calling the physician, from the violence of the disease, and some die immediately after calling him, having lived, perhaps, only one day or a little longer, and before the physician could bring his art to counteract the disease; it therefore becomes necessary to know the nature of such affections, how far they are above the powers of the constitution [tōn sōmatōn],[10] and moreover if there is anything divine in the

[7] *Geschichte der Medicin*, 3rd edit., III, 428.

[8] *Bul. de la Soc. Franç. d'Hist. de la Méd.*, tom. xxvii, nos. 3 et 4, mars-avril, 1933, p. 77.

[9] Haeser, (*loc. supra cit.*), suggests that these were cases of diphtheritic paralysis, but I do not agree with his suggestion.

[10] Jones's translation, "the strength of men's bodies," is much better.

diseases, and to learn a foreknowledge of this also." Adams has the following note on this passage: "It has puzzled all the commentators, ancient and modern, to explain satisfactorily why Hippocrates in this place seems to adopt the popular creed, and acknowledge that a certain class of diseases are of a divine origin, whilst in his treatises 'On Airs,' etc., and 'On the Sacred Disease,' he combats this doctrine as being utterly unfounded." Now I venture, with all diffidence, to suggest that Adams overlooked one point, and that is that Hippocrates was not stating as a definite proposition that there was something divine in diseases, but was suggesting that the physician should try to find out whether there was anything divine in them. He is really asking the question, Is there anything divine in diseases? He does not, however, answer it in the *Prognostic*. That treatise is full of excellent advice on the formation of a prognosis from a consideration of the symptoms and signs presented by the patient and his body, but there is not a word which will enable the physician to detect the divine element. I submit that this silence may be fairly interpreted to mean that Hippocrates did not believe in it.

In the latest English translation of Hippocrates, that by Mr. Jones in the *Loeb Classical Library*, in which the Greek text is given with the English version, the sentence containing the words *the divine element* tŏ thĕiŏn, is omitted from both versions, and the translator has appended the following footnote: "The clause omitted by Kühlewein 'and at the same time whether there is anything divine in the diseases' is found in all MSS. It is contrary to Hippocratic doctrine." By those, however, who are of the opinion that the clause should be retained because it is found in all the MSS., it may be argued that as Hippocrates advised physicians to inquire whether there was anything divine in diseases, there was in his opinion some divine element or factor to be looked for, at any rate in some diseases. There is force in this argument, to which, however, it may be replied that what Hippocrates meant was that while there was a popular belief that there was something divine which plays a part in the nature of a disease, he did not think so himself; still, there was no objection to an inquiry into the truth or falsehood of the belief. Hippocrates, however, himself answers the question in no uncertain terms in the treatises *Airs, Waters, Places* and the *Sacred Disease*. The argument is, briefly, that certain diseases alleged to be of divine origin are no more of such origin than are other diseases. Either no disease is derived from the gods or all are. It is quite clear that Hippocrates had no belief whatever in a special, divine factor in the causation of diseases. And whatever view as to its genuineness may be taken of the passage in *Prognostic*, it is also quite clear that he never invoked a "something divine" as a causation either of epidemics or of the severity of febrile or other diseases.

The reader who expects to find in the books entitled *Epidemics* any explanation of the rise and fall of an epidemic, will be disappointed. The writer of *Epid.* I and III describes the weather which occurred during three different periods—and each of these states of the weather he termed the "constitution" of the period—and then he gives an account of the diseases which were prevalent during each "constitution." He leaves you to infer that the diseases were due to the weather conditions. In *Airs, Waters, Places*, however, we find a definite opinion, for it is distinctly laid down that the diseases which will be endemic or epidemic in any given city or town will depend upon local conditions, to wit, the prevailing winds, aspect, nature of the soil, character of the water and the modes of life and customs of the inhabitants. The changes of the seasons and the risings and settings of certain stars and constellations must also be taken into account. Further, the illnesses suffered by any individual will be determined by his habits. The writer gives a list of diseases which are to be found in cities especially exposed to hot and cold winds respectively, and also in those otherwise situated. Changes in diseases are particularly likely to be associated with sudden changes in climatic conditions, and with

the risings of Sirius and Arcturus and the setting of the Pleiades. In *Sacred Disease*, xvi, a brief account is given of the action of the winds, and especially the north and south winds, on the atmosphere and the human body. Epilepsy occurs most frequently when the south wind blows, then when the north wind, and lastly when the others. It is not suggested, however, that there are epidemics of epilepsy.

It is to be observed that Hippocrates does not make use of the combination of words "epidemic constitution," but writes of "constitution" (katastasis), only. He usually employs the term in respect of states of the weather, but occasionally also of disease. I do not know who first used the combination. It is found in certain writings of Nicolas Massa (1540), and Réné Moreau (1646). But Sydenham (1676) is chiefly responsible for its dissemination in epidemiological literature. None of these writers defines what he means by the term. By some authors it has been confused with *ti thĕiŏn*, a something mysterious which causes epidemics. Personally I have no belief in any such factor. However, as I have discussed the subject at length elsewhere I will not pursue it further here.[11]

It is in the *Breaths* that we find a real attempt to explain an epidemic. After endeavouring to show that air[12] in its various forms is of the utmost importance generally in the economy of nature, the writer goes on to say (chap. v and vi), that he "will show that diseases are all the offspring of air."

"I will begin in the first place with the most common disease, fever, for this disease is associated with all other diseases. To proceed on these lines, there are two kinds of fevers; one is epidemic, called pestilence [lŏimos], the other is sporadic, attacking those who follow a bad regimen. Both of these fevers, however, are caused by air. Now epidemic fever has this characteristic because all men inhale the same wind; when a similar wind has mingled with all bodies in a similar way, the fevers too prove similar. But perhaps someone will say: 'Why then do such diseases attack, not all animals, but only one species of them?' I would reply that it is because one body differs from another, one air from another, one nature from another, and one nutriment from another. For all species of animals do not find the same thing either well or ill-adapted to themselves, but some things are beneficial to some things and other things to others, and the same is true of things harmful. So whenever the air has been infected with such pollutions as are hostile to the human race, then men fall sick, but when the air has become ill-adapted to some other species of animals, then these fall sick."

In this paragraph it is recognized that an epidemic fever is due to a common cause, namely, the air; but the same air does not attack all species of animals; there are different airs and different bodies of animals. Further, there is a hint that it is not the air so much as something that has polluted it that is the cause of an epidemic.

It is true that these statements are not backed up by a single particle of evidence. The writer may be putting forward his own opinions or quoting those of others. The following passages in the *Nature of Man*, ix, lead me to think that he may have been quoting a perhaps commonly held opinion: "Diseases arise, in some cases from regimen, in other cases from the air by the inspiration of which we live. The distinction between the two should be made in the following way. Whenever many men are attacked by one disease at the same time, the cause should be assigned to that which is most common, and which we all use most. This it is which we breath in. . . . But when diseases of all sorts occur at one and the same time, it is clear that in each case the particular regimen is the cause. . . . But when an epidemic

[11] *Proc. Roy. Soc. Med.*, 1927, xxi, 119 (Sect. Epid. 1,), and *A Text Book of Infectious Diseases* (1928), Chap. I.

[12] The Greek writer explains that *phusa* is air, *pneuma*, within the body, and *aēr* is *pneuma* out of it. Jones translates *pneuma* by "wind," *phusa* by "breath," and *aēr* by "air." But the Greek writer is by no means consistent in his use of the words.

of one disease is prevalent, it is plain that the cause is not regimen but what we breath, and that this is charged with some unhealthy exhalation."

A large portion of the collection of fragments known as the *Nature of Man* is devoted to showing that the quantity of each humour in the body varies with the season of the year, and that as the seasons vary in character from one year to another so does the quantity of the humours. As diseases are caused by excess or diminution of the quantity of the humours, so they vary with the character of the seasons.

The variation of diseases with the seasons is pointed out also in *Humours*, xiii, where it is explained that a normal season produces diseases normal to the season, but an abnormal season produces diseases abnormal to the season but normal to the character of the abnormal weather. Thus a wintry spring gives rise to the diseases peculiar to winter. Besides seasonal variation "account must also be taken of the condition of the body when the seasons come upon it." Indeed, from a close study of the body and its diseases, "as it is possible to infer diseases from the seasons, so occasionally it is possible from diseases to forecast rains, winds and droughts." (*Humours*, xvii.)

Aphorism III, xix, states that "all diseases occur at all seasons, but some diseases are more apt to occur and to be aggravated at certain seasons." There are several other interesting epidemiological statements in this Section of the *Aphorisms*.

From a perusal of the Hippocratic Collection and especially of the *Epidemics*, in which more details are given of febrile diseases than elsewhere, I am led to conclude that during the fifth to third centuries B.C. the countries bounding the Ægean Sea were most unhealthy and were infested with fatal, febrile diseases of microbic origin, as well as with malaria. The physicians of those regions and of that age differentiated between the acute febrile diseases to a much greater extent than they have usually been given credit for. They also recognized not only the endemicity but also the epidemicity of certain febrile diseases. Further, they observed seasonal incidence, and endeavoured to explain it by the interaction of the bodily humours and the states of the weather. They recognized that there was more than one factor in the causation of these diseases, and in the case of epidemics they had a shrewd suspicion—which to one or two of the more dogmatic minds amounted to a certainty—that there were other factors at work besides the interaction of the humours and the weather, and especially some pollution or unhealthy state of the air—what was in later ages called a miasm.

Section of Medicine

President—Sir FARQUHAR BUZZARD, Bart., K.C.V.O., M.D.

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DISCUSSION ON THE PRESENT POSITION OF COLLAPSE THERAPY IN LUNG DISEASE

Dr. Rist (Laennec Hospital, Paris).—Twenty-one years have elapsed since, on the suggestion of the late Sir William Osler, I wrote a somewhat elaborate critical review on artificial pneumothorax.[1]

Although Forlanini's original papers date back as far as 1894 and John D. Murphy's independent contribution as far back as 1898, and although Brauer had published his first few cases in 1905, the topic of induced pneumothorax as a rational treatment of lung tuberculosis was in 1913 a comparatively new one. The procedure so far from being universally accepted and approved, met with considerable reluctance and even opposition. The English and French literature concerning it was scarce: my personal experience of the technique and results of collapse therapy was ludicrously short—I had begun treating my first cases only in 1911. Looking back to this juvenile paper of mine, I feel that my undertaking to write it might well have been criticized for its rashness. Nevertheless it received Sir William Osler's approval, and I think it gave a fairly truthful account of the prevailing opinion among medical men practising artificial pneumothorax.

When therefore you invited me to open a discussion on the present position of collapse therapy of lung disease, I naturally turned back to my twenty-one year-old paper as a convenient basis for measuring the distance we have travelled and comparing the present with the past.

Let us first consider the technical improvements. Our instrumentarium has hardly been modified. The trocars, the needles, the apparatus for introducing gas into the pleural cavity in measurable quantities and under measurable pressures are practically the same as were used a quarter of a century ago. But their handling has become more familiar to us; we are more accurate in our measurements. The most dreaded initial accident, gas embolism, has become so exceptional that it is almost negligible. The caution which I advocated in 1913, and to which I still adhere, of beginning the first filling with pure oxygen, has perhaps contributed to this increase in safety to some extent. The use of nitrogen is no more insisted upon. Atmospheric air is just as efficient, its absorption being only by one-tenth shorter than that of nitrogen.

But we have had the benefit of more decisive technical improvements. In the first place I should like to mention the thoracoscopic cauterization of adhesions for which we are indebted to Jakobaeus (Stockholm).

In a large proportion of cases we were unable to achieve a perfect collapse because the most diseased part of the lung remained uninfluenced owing to one or several adhesions keeping a cavity patent. The patient, even when his general condition had been bettered and the amount of his sputum not inconsiderably

[1] *Quart. Journ. Med.*, 1912-13, vi, 259.

reduced, was in permanent danger of reinfection in the contralateral lung. A complete and definite recovery was out of question. The Jakobaeus method has entirely changed the outlook of such cases. It has made gratifying successes of what would have been sad failures. It deserves certainly to be made use of more freely than it is at present.

One serious menace to the favourable end-result of an artificial pneumothorax successfully initiated was the development of large flat symphyseal adhesions extending upwards from the base, generally after an effusion. No increase in gas pressure was capable of preventing gradual closure of the pleural cavity, and consequently premature interruption of the therapeutic collapse. It is true that in a few patients the process of recovery by pneumothorax goes on steadily, regardless of lung re-expansion. But this is exceptional. Usually, premature re-expansion has as its natural consequence reopening of cavities, spreading of lesions and recurrence of symptoms. Now we are in some measure able to overcome this danger by substituting sterilized paraffin oil for gas in the pleural cavity. This, I think, is the true and really useful indication for oleothorax, first proposed and put into practice by my countryman Bernou. Oleothorax as a treatment for purulent pleural exudates or as a method for improvement of imperfect lung collapse, has been something of a disappointment. But as a method for preserving the successful collapse of the diseased upper lobe against impending adhesion I have found it extremely useful.

Perforation of the collapsed lung was regarded as a complication, almost invariably fatal. Although it is still a severe accident, often terminating in death, we are not to-day rendered altogether helpless by its occurrence, and further, we can do something to prevent it. The rupture frequently takes place in the immediate neighbourhood of the lung-insertion of a string-shaped adhesion, the pull of which acts directly on the diseased tissue. By destroying the adhesion with the thoracoscope we avert the probability of perforation. In institutions where Jakobaeus' method is commonly in use, perforations have certainly become less frequent. But even when the rupture has occurred we can sometimes avoid its awkward consequences. Continuous oxygen-breathing by means of a nasal catheter helps the patient to pull through. A needle provided with a valve allowing the gas to be expelled from the pleural cavity during expiration and prohibiting its entrance during inspiration has been devised by Cardis (Leysin); inserted and kept permanently through the chest wall it relieves the mediastinum from over-pressure, prevents the danger of asphyxia, and in many a case has kept the patient alive during the initial phase, which is the most dangerous. Later on, the rupture, provided the opening is not too large, may heal spontaneously, the lung being at rest; the open or valve pneumothorax is converted again into a closed one, and the collapse treatment can be maintained by regular refills.

Side-by-side with the technical advances which have made artificial pneumothorax so much more efficient than it was formerly, other methods of collapse have been evolved, requiring a close co-operation between the physician and the surgeon. Whenever an artificial pneumothorax remains imperfect and inefficient because of adhesions which the Jakobaeus method cannot destroy, or whenever pneumothorax is impracticable on account of extended symphyseal adhesions, the question arises whether we are to resort to phrenic avulsion or to thoracoplasty or to both.

I do not intend to dilate upon those surgical methods, which have so widened the scope of collapse therapy, but, regarding phrenic avulsion, I should like to remark that it should not be considered exclusively from the point of view of collapse. It came as a surprise to most of us to find that apical lesions were influenced favourably, and even definitely healed after phrenicectomy, just as well as cavities situated at the base of a lung, in the immediate neighbourhood of the diaphragm. Furthermore, we are all aware that the curative influence of phrenic

avulsion cannot be measured by the height to which the paralysed diaphragm ultimately reaches, or, in other words, by the degree of lung volume reduction. The height of the lung may be diminished by one-third or even more, and the lesions remain unchanged, whereas in other cases we see cavities closing and disappearing within a surprisingly short time after a phrenic avulsion which produces hardly any change in the position of the diaphragm. There is still much that is unknown and mysterious in the functions of the phrenic nerve. Certainly when we cut it we do more than inhibit its motor action on the diaphragm. Luciani's experimental studies suggest an influence of the phrenic nerve on the smooth musculature of the broncho-pulmonary apparatus by way of anastomotic connexions with the sympathetic system.

Thoracoplasty, on the contrary, is strictly a method of collapse therapy. Its technique has improved constantly. The operation has lost most of its terrors, and can boast an increasing proportion of excellent and permanent successes. It has saved many lives and restored many a bed-ridden patient to normal activities.

Having now reviewed the various methods of collapse therapy which are at our disposal to-day, let us consider what position they hold or, perhaps I should say, ought to hold—at the present time, in the treatment of lung disease, and, primarily, of lung tuberculosis.

I suppose we all agree that the diagnosis of pulmonary tuberculosis should be founded not on general and functional symptoms only, but on the ascertainment of definite physical signs—among which the alterations revealed by X-ray pictures are indispensable and all-important—together with the presence of tubercle bacilli in the sputum.

Now, given a case of lung tuberculosis thus defined, what shall determine our choice of collapse-therapy? Or, to put the question more bluntly, we have to choose between collapse-therapy and . . . what else? Twenty years ago, one would still have heard many voices in favour of tuberculin. To-day, the mention of tuberculin as a treatment of consumption raises almost no echo, and justly so, I feel sure. Tuberculin has been given a prolonged and fair trial. As a routine therapy of definite lung tuberculosis, it has failed. Even the faithful few who still adhere to it would not dream of comparing its results with those of artificial pneumothorax.

To-day, some supporters of gold-therapy would perhaps take up the challenge. The specific influence of gold salts on tuberculous lesions is still under discussion. The great hopes which the first announcements of the Danish school raised have been followed by great disappointments. It seems as if sanocrysin and other gold preparations were useful in a certain proportion of cases, but what this proportion may be, nobody can tell. Certainly the failures outnumber the successes; very few successes are complete and fewer still are lasting. Certainly, too, gold salts are not harmless. The position of gold, all over the world, is strangely critical just now, but I feel confident that its prestige will sooner be restored as a monetary standard than as a specific therapeutic agent.

What then, if we discard or postpone collapse, shall our alternative be? I can see only one: namely the time-honoured air-and-rest cure, realized at its best in a sanatorium. It has behind it a long and valuable record of curative accomplishments. It is a rational, common-sense method, physiologically well founded. It is a basic method. Even if we had at our disposal a specific treatment of tuberculosis which would equal in efficacy arsphenamine against syphilis or antitoxin against diphtheria, we should, I feel certain, be under the strict obligation of applying it under conditions of perfect rest.

But I have serious doubts as to the soundness of the still widespread opinion, that sanatorium treatment should be regarded as an alternative to collapse therapy. I would rather say that the most favourable conditions for establishing a successful

pneumothorax, or for deriving the greatest possible benefit from a phrenic avulsion or a thoracoplasty are to be found in a sanatorium.

I confess that I cannot agree with the advice so often given: "try sanatorium treatment first; if after six or twelve months, the rest-cure has not restored your health, we will perform a pneumothorax." One obvious objection to this scheme is that it promises more than it can be certain to perform. Pneumothorax may be quite easy to day but after six months of inefficient rest-cure, the pleuræ may be found adhesive and collapse unobtainable. By undue postponement of the best form of treatment, our patient may have lost his best chance of recovery.

There are many physicians nowadays who will readily admit the advisability of employing collapse therapy without delay, whenever there is a cavity present; but they are still reluctant to use it immediately for small active lesions, when there is no cavitation and the symptoms are slight. Those cases, they maintain, do marvellously well with sanatorium treatment. It has often happened that I have sent such a patient, with a few dark spots in one apex, a history of a recent hæmorrhage, hardly any sputum and only scanty bacilli, to a sanatorium, with the request that a pneumothorax should be induced. Later, I have received a letter from my sanatorium confrère stating that the improvement had been so rapid with the air-and-rest cure that he had thought it wise to abstain from pneumothorax treatment. As a matter of fact, all the symptoms had subsided and the X-ray picture showed an almost complete disappearance of the condensed spots. No doubt some of these cases were definitely and permanently healed. But a large proportion had a relapse soon after they had returned to work; the relapse was almost invariably more severe than the first episode. Had pneumothorax been performed at once, how much time, sickness, anxiety and money would have been saved!

I have had personal experiences enough of that kind to become convinced that even in slight recent cases, there is absolutely nothing to be gained by postponing pneumothorax. I have no hesitation in asserting that tuberculous lesions of the lung are healed more definitely and more lastingly under collapse combined with a rest-cure than under a rest-cure only. Moreover there are many tuberculous lesions of the lung and common ones, lobar involvements with cavitation for instance, which could hardly ever heal with sanatorium treatment and which usually heal with collapse. The progressive readaptation to normal life and work is easier, fraught with much less danger with collapse than without it. Also the process of readaptation takes place under the permanent control of the physician, because of the unavoidable periodical refills. Furthermore, complications such as effusions are seldom observed in the course of a pneumothorax undertaken for incipient cases. The treatment generally runs its whole course smoothly and the capacity for work is rapidly restored.

I was glad to find myself in full agreement on this point with several of my British colleagues. A remarkable paper by Dr. Cedric Shaw published in the *Quarterly Journal of Medicine*, 1933 (N.S. ii, p. 179) strikes, in my opinion, exactly the right note, and the statistical tables which it puts before the reader in order to compare the results of sanatorium treatment with those of early pneumothorax are most illuminating.

The sooner pneumothorax is attempted, the greater are the chances of a free pleural space and a complete collapse. But a pleural effusion may have preceded, several years before, the development of tuberculous lesions in the lung, and there are also cases in which pleural adhesions are almost contemporaneous with the lung lesions. The same reasons which speak against the postponement of pneumothorax speak also against the postponement of phrenic avulsion when pneumothorax has been proved impossible. We often see that between the unsuccessful attempts to create a pneumothorax and the performance of a phrenic avulsion, several months, or even a year or two, have been allowed to elapse. I cannot find any justification for such a delay. Once it has been determined that in a given case collapse therapy

is the right thing to employ, then—failing the most efficient way to realize it—the next best method must be adopted—I should like to say, almost automatically.

The favourable results of phrenic avulsion are sometimes so rapid and so striking that a controversy has arisen during these last two years as to whether it is not advisable to prefer it to pneumothorax which would be employed only after an eventual failure of phrenicectomy. Morin (Leysin) has been the most zealous advocate of this scheme, arguing that it would do away with the so-called "obnoxious slavery of periodical refills," and give the patient more freedom. He added, that if phrenic avulsion proved unsuccessful, one could always have recourse to pneumothorax. I think, for my part, that Morin's proposal is unsound. The choice being between artificial pneumothorax and phrenic avulsion, the latter will always be available if we begin by the former and it fails. But if we begin with phrenicectomy, we do not know whether, after it has proved unsuccessful, pneumothorax will still be possible. Adhesions may have developed during the delay.

Moreover, the advantages claimed by Morin for phrenicectomy, namely the greater freedom given to the patient, obvious as it may seem at first sight, is, if we go a little deeper in the matter, a dangerous delusion. The so-called slavery of periodical refills is really one of the most precious safeguards of the patient's recovery, because it means the permanent control by the physician of his conduct, of his way of living, and of his healing process. After a phrenic avulsion patients are only too prone to escape the physician's control. They rely with too much optimism and self-confidence upon the immediate, often strikingly good, results of the operation, return to work earlier than they should, and fall victims of a relapse.

I have tabulated recently my 200 first hospital cases of phrenicectomy ranging from October 1923 to June 1931. If we consider the results six or eight months after the operation, we find that in 100 (50%) of those cases, they are excellent and seem to point to a prompt and lasting recovery. But surveying again our patients in November 1933, after a period of two and a half to ten years following the phrenic avulsion we find that the number of definite recoveries is only 26 (13%). If we add 16 cases of improvement (not equivalent to recovery) our ultimate favourable results amount to 42, or 21% only, whereas our failures number 138 (69%) including 86 deaths (43%). Twenty patients (10%) have been lost sight of.

It is, I think, interesting to remark that out of our 100 immediately favourable cases, 79 had had the benefit of sanatorium treatment lasting from six to thirty months after the operation. It is amongst those 79 that almost all our recovered patients (23 out of 26) and our improved patients (15 out of 16) are to be found. Such figures are a sound argument in favour of the combined sanatorium and collapse therapy.

The tendency to-day is evidently—and, in my opinion, very fortunately—to perform pneumothorax, or, failing pneumothorax, phrenicectomy—not only in severe cases, but in mildly active cases, not only for extended and excavated lesions, but for circumscribed non-ulcerous lesions. We should use it not only as a last resource after an unsuccessful and prolonged trial of other methods, but as soon as possible after the initial onset, because it is the best, safest, and shortest method of treatment. When pneumothorax and phrenicectomy, or, as may sometimes prove advisable, a combination of both prove inefficient, no time should be lost, but the opportunity for a thoracoplastic operation should be seized upon without delay if it can be undertaken with real chances of success.

This applies, of course, essentially to unilateral tuberculosis of the lung. But, regarding pneumothorax and phrenicectomy, we do not feel any longer obliged to submit blindly to the rule of strict unilaterality. When the method was still, so to speak, in its experimental stage, when the healing influence of collapse had to be demonstrated to a timid, conservative and incredulous medical profession, the rule was a wise one. I still consider it a golden rule for beginners. But physicians

who have acquired experience in collapse therapy may be permitted to take some well-advised liberties with the law. We all know that a successful pneumothorax on one side often has as its consequence the disappearance of more or less slight lesions on the other side. It seems as if by mechanically helping one diseased lung to recover, we increase the resistance of the whole body to tuberculous infection and stimulate the healing process in the non-collapsed lung. Therefore a not inconsiderable proportion of patients who would formerly have been excluded from the benefit of collapse therapy on the ground that their disease was not strictly unilateral, are to-day given a chance, and have no reason to regret it.

Furthermore, there are certain types of bilateral tuberculosis of the lungs which we are now in position to improve and even to heal permanently by collapsing both lungs simultaneously. When Parry Morgan suggested for the first time, twenty years ago, the feasibility of bilateral pneumothorax he did not meet with much encouragement or approval. It seemed foolhardy, an impossible proposition; violating the most sacred principles of sound physiology. It is now legitimately practised all over the world. A bilateral pneumothorax is a delicate undertaking, requiring a good deal of skill and care and as much perseverance and determination on the patient's as on the physician's side, but it has proved an invaluable extension of the possibilities and achievements of collapse therapy.

We must keep in mind also to what a remarkable extent collapse therapy has made it possible to treat conditions which lung tuberculosis, as a complicating factor, used to render hopeless. Think of such awkward combinations as consumption and diabetes, which were rightly regarded as fatal. We may to-day not unreasonably endeavour to treat both diseases in the same patient; the association of pneumothorax with insulin has often done wonders. To operate on an appendix, on a gall-bladder or on a gastric ulcer was a serious risk if the patient had an active, or even an arrested, lung-tuberculosis. If the diseased lung is under the protective influence of an artificial pneumothorax, the danger is considerably lessened. The bearer of an artificial pneumothorax may have a tuberculous cæcum or a tuberculous kidney removed, and recover. Tuberculous laryngitis complicating lung tuberculosis heals spontaneously after the diseased lung has been collapsed successfully. The aggravating influence exerted by pregnancy and confinement upon the course of consumption is often inhibited by artificial pneumothorax and we may safely allow a woman successfully treated by lung collapse to bear children.

The striking efficacy of pneumothorax and its surgical substitutes in the treatment of tuberculosis of the lung has naturally led to an attempt to treat other diseases of the lung by the same methods, more especially abscess of the lung and bronchiectasis. In a paper recently published in the *Lancet*, Dr. L. S. T. Burrell,[1] a strong supporter of the view that artificial pneumothorax should be done early, mentions eight cases of lung abscess treated in 1923 and in 1924 by collapse therapy in seven of which the patients were well in 1933. His figures for bronchiectasis are not quite so encouraging. Two patients only out of eight are well. The figures which I gave a year ago at the Montreal meeting of the American College of Physicians represent all the cases of bronchiectasis observed in my department at the Laennec Hospital during the period from 1920 to the end of 1931.

Their total number is 90, but only in 59 cases was artificial pneumothorax attempted. We failed to secure it in 22 cases, the pleura being totally or extensively adherent. Among the 37 patients whose lung could be successfully collapsed 17 were not benefited by it, for various reasons. But 20 patients, one-third of the total, derived the greatest possible benefit from pneumothorax treatment. They were rapidly freed from all morbid symptoms; their discharge disappeared; the cough ceased; fever and loss of weight stopped; they were to all appearances restored to health. Moreover, in 11 of my cases of bronchiectasis in which adhesions had

1 *Lancet* 1933 (ii), 1414.

caused the failure of pneumothorax, phrenicectomy was performed: three patients made a perfect, complete, permanent recovery; four other patients were greatly improved but not cured. I feel pretty certain that if bronchiectasis were always diagnosed early and if the pneumothorax treatment were always initiated as soon as the diagnosis had been established, the proportion of recoveries would increase substantially.

In conclusion I would say that, with regard to lung tuberculosis, collapse therapy holds a dominant position. It seems most likely that the majority of patients suffering from lung tuberculosis have been, are, or will be, some time good cases for pneumothorax. It is our duty to seize the opportunity and not to let it pass unused. Collapse therapy has completely altered the outlook of lung consumption. It has changed it from an incurable to a curable disease. Recoveries were formerly the rare gifts of exceptional good luck; they are now normal achievements, such as can be expected from a logical, scientific therapy.

If collapse therapy were merely one of many methods, we should not observe as we do, its unceasing development—the various improvements which, year after year, contribute to make it more effective, and to procure its benefits in cases which were formerly beyond its scope. Indeed, Forlanini was a great man.

Dr. R. C. Wingfield: I think that now, after many years' experience of artificial pneumothorax treatment, we are wise to take stock of its results and to endeavour to estimate its value in the treatment of pulmonary tuberculosis. I do not think that we can get this information by comparing the survival rates of large numbers of patients who have been treated by artificial pneumothorax with the survival rates of those who have not been so treated. Such figures can never be of much value as guides for the correct treatment of the individual case, for two reasons: firstly, our classification of pulmonary tuberculosis is too broad, and artificial pneumothorax is used for so many different types of cases and for so many different reasons that it is impossible to get really comparable control groups with which to check our figures: secondly, patients who have had artificial pneumothorax treatment have pulmonary tuberculosis just as surely after the collapse as they had before it was undertaken, and, although some of their individual risks may have been altered or removed, they are, taken in bulk, still exposed to exactly the same risks as the average consumptive, and therefore their survival rates will be approximately the same as that of the average case. For these reasons it is the individual case that we must study if we are to compute the value, and the shortcomings, of this particular therapeutic method.

Apart from individual and peculiar cases in which artificial pneumothorax is undertaken to meet special social, financial, or psychological conditions, there are two main reasons for its use.

(1) To secure rest of the lung—exactly as a splint is used for a limb—to promote natural healing of active tuberculosis, ordinary routine methods having been unsuccessful.

(2) To obtain the complete collapse of a lung that has been so structurally damaged by cavitation and fibrosis that its expansion is a constant source of danger: in fact, to produce as nearly as possible the effect of a total or partial excision of lung.

Now in both types of case the actual success or failure of the treatment depends upon the behaviour of the visceral pleura, and we appear to have no adequate means of controlling the behaviour of this tissue. Dealing with the position from this point of view in both types of cases I have mentioned, in type No. 1 it may be assumed that there is not much structural damage of the lung, but in cases in which the activity of the tuberculosis, as judged by clinical and radiological criteria, is obstinate, artificial pneumothorax offers to the physician a tempting way out of his difficulties

and impatience sometimes leads him to subject his patients to a treatment that in a definite proportion of cases does permanent structural damage to the lung—in fact, produces that very condition for which we prescribe collapse therapy in other cases. I will illustrate my point by two cases.

I. This patient, after three months complete rest under sanatorium conditions, still showed evidence of activity of the disease in his right lung. An artificial pneumothorax was successfully induced. Within a short time fluid appeared in the pleural cavity with its usual increase of intrapleural pressure, an adhesion snapped under the strain, the lung was torn, and a streptococcal pyopneumothorax with a permanent pleuropulmonary fistula resulted.

II. In this case a right pneumothorax was induced for obstinate activity. The immediate result was excellent and the patient was able to return to work. Pleural involvement, however, occurred and her working periods were interrupted by this and by the various attempts at its treatment. She finally returned to the sanatorium with a grossly fibrosed and cavitated right lung producing so many symptoms and so much disability that life was unbearable. However, further collapse by thoracoplasty successfully dealt with the condition.

Now both these cases show grave and dangerous structural damage directly due to pleural involvement—which, in its turn, was directly due to artificial pneumothorax treatment—and such cases are by no means rare. As a converse, I may show the perfect after-result, with the lung fully expanded and the lesions healed, the pleura having remained without involvement.

I do not suggest for one moment that we are to be deterred from using artificial pneumothorax in suitable cases because of possible, but rare, complications—though pleural involvement is not so rare—but I do suggest that these possibilities must be given full weight.

The institution physician, who is usually responsible for the artificial pneumothorax, is tempted to form his conclusions too much on small cross-sections of the disease. The patient has active disease, collapse is used, activity ceases, and the immediate result is excellent. But pulmonary tuberculosis is a chronic disease and the artificial pneumothorax which has saved so many lives may later actually be the cause of dangerous illness.

At present we seem entirely unable to control pleural behaviour. Many suggestions have been made—the use of small versus large needles, slow refilling, warming and sterilization of introduced air, etc. have been tried without consistent results, and still our patients seem to be at the mercy of the whims of this inconsequent tissue, which either cuts short our time of splinting and draws out to the thoracic wall a distorted lung containing foci of active disease, or else ultimately leaves us with a lung in such a condition that we have to turn to the surgeon to clear up the mess that we have produced. This problem of pleural behaviour demands closer study, and until we have learned to control it, collapse by artificial pneumothorax in a relatively slightly damaged lung should be adopted with caution. In this connexion would it not be possible to explore further the possibilities of paralysis of the diaphragm accompanied by a longer period of rest? This point of view is set out in a spirited manner by Dr. Morin in his M.D. thesis, "Pneumothorax *versus* Phrenicotomy." He definitely champions paralysis of the diaphragm as superior to the artificial pneumothorax—an interesting suggestion, but one that needs careful consideration before it can be accepted.

In type No. 2 the position is somewhat different. Here the physician is confronted with a permanently damaged lung which must be an increasing source of danger to the patient. Action must be taken, and the usual procedure is to produce collapse by an artificial pneumothorax aided by section of adhesions if necessary, and/or by phrenic evulsion, and, if these are unsuccessful, by thoracoplasty. And here let me interpolate that if a collapse by artificial pneumothorax is deemed necessary and the condition of the contralateral lung permits it, then if a complete

or efficient pneumothorax is impracticable the case is suitable for thoracoplasty or pneumolysis and plombage.

In cases in which a complete and efficient pneumothorax has been produced, we are, however, still at the mercy of the pleura, but in rather a different way. Such a lung must never be allowed to re-expand fully, and therefore pleural involvement with effusion leading to obliteration of the pneumothorax by gross pleural thickening, retraction of the hemithorax, and displacement of the mediastinal contents with the minimum of lung expansion, may mean a successful termination of the case. On the other hand, there is still the danger of lung rupture in a small, but definite, percentage of cases, and the effusion, whether secondarily infected or not, is not always benign. It may point through the chest wall, or it may be obstinate in causing ill-health in spite of every form of treatment. When these accidents occur, attempts have been made to put them right by thoracoplastic operation—with conspicuous ill-success. If they do not respond to pleural lavage, the outlook is indeed black. Again, we see that the pleura commands the situation, and therefore, since we are agreed that such a lung should be permanently out of action, and considering the great advance in the technique of thoracoplasty in recent years, we ought to consider whether it would not be wiser to use thoracoplasty for this type of case in the first instance, instead of dallying with an uncertain artificial pneumothorax.

To sum up: I am convinced of the supreme value of collapse therapy in general, and of artificial pneumothorax in particular, in the treatment of pulmonary tuberculosis, but I am sure that where there is little structural damage to the lung pneumothorax should not be undertaken until the evidence for its necessity is incontrovertible, and in cases in which there is gross structural damage, the possibility of thoracoplasty instead of pneumothorax, as the less dangerous procedure, should be carefully considered.

Dr. S. Vere Pearson: I shall limit my remarks to the treatment of chronic pulmonary tuberculosis. Reviewing the position now as compared to twenty years ago, there are three striking changes: (1) we have learned to utilize collapse therapy oftener; (2) the methods of compressing the lung have developed and are developing; (3) dependence upon X-ray findings is greater than it used to be. The percentage of cases in an ordinary sanatorium or similar institution in which artificial pneumothorax is attempted, has risen by degrees from something like 10% to something over 40%. At Mundesley to-day with 65 patients 73% are having or have had collapse therapy. This is probably a high percentage, for the reason that I was responsible in August 1910 for the first artificial pneumothorax induction in England. Nearly all the advances which have come about during the last twenty years, for the most part in connexion with the selection of patients, and of the particular procedure to adopt and the best time to do it, are due to improvement in the technique and greater use of radiology. None the less, there is a tendency to treat patients too much on the X-ray appearances alone. These are only part of the picture; however important, they must not be depended upon too exclusively: they must be taken strictly in conjunction with the clinical condition.

There are still many parts of the country in which too little is known about compression treatment. Even where tuberculosis officers are readily available, and still more amongst general practitioners, sometimes no experience of artificial pneumothorax is to be found. There are some tuberculosis officers without artificial pneumothorax apparatus, and even some who suffer under the disability of having no easily available X-ray facilities. A year or two ago the Joint Tuberculosis Council published a short memorandum from a committee formed by them, of which I was convener. In this memorandum it was pointed out that the public

tuberculosis schemes should provide more time and more personnel in order that collapse therapy should be available for the benefit of patients to an adequate extent. Despite all efforts, I could name several large towns in this country (cities with about 100,000 inhabitants) where no one can be found to practise collapse therapy, or where no one other than the tuberculosis officer is competent to give a refill, and he often has to do this in circumstances which are not altogether acceptable to private patients.

I have already stated that at Mundesley we have 73% of a total of 65 patients having collapse therapy. We have three doctors attached to the sanatorium and they are kept fairly busy. The amount of extra work thrown upon the doctors at a sanatorium, compared with that of ten years ago, is very great. I think the local authorities and even those at the Ministry of Health who supervise these matters do not fully realize the extent of this increase. If one compares Mundesley, where three doctors are not idle, with an institution of perhaps four times the size, it will generally be found that there are perhaps only four doctors in such an institution, and there are many with about 100 patients who have only two doctors. It is impossible for the medical officers of institutions, or for tuberculosis officers, to cope with the work entailed in the modern methods of treatment of pulmonary tuberculosis, even if they have the necessary experience and skill, unless they have more help. This can sometimes be clerical, instead of medical, assistance, but more doctors are wanted to cope with the modern methods of treatment.

Though collapse therapy is still not used sufficiently in many quarters, it is nevertheless true to say that in some places it is overdone; not so much that it is used too early as that too much reliance is placed on it. An artificial pneumothorax when feasible, is excellent treatment for most one-sided cases; and though every case must be taken on its merits, it is probably true that the mistake of resort to artificial pneumothorax too late is commoner than the opposite one. The good results of collapse therapy should not, however, blind doctors and patients to the necessity for a careful way of living; in other words, the collapse therapy must be superimposed upon a sanatorium régime. The most important factor of this régime is rest, and a due amount of rest in bed must always be imposed.

Recently I have been reviewing the late results of simple artificial pneumothorax treatment applied at Mundesley from eighteen to twenty-three years ago. Though many have died from tuberculosis a number of these patients are alive and well to-day; a few have died of old age or from non-tuberculous causes. What has struck me in making this review is this—that several more might well have been alive and enjoying good health to-day had they been more skilfully treated; a patient in whose case artificial pneumothorax was induced in August 1910, e.g. made a good recovery but after he had left my care a doctor, finding opalescent fluid in the chest, inserted a tube. Another of my early cases abandoned the artificial pneumothorax upon the advice of the late Dr. A. Latham (few knew better in those days) after only fifteen months, led a strenuous ordinary life, relapsed and died. Another who lived in good health, though originally desperately ill, for over seven years, died, as a result of bursting an enormous collection of fluid under the skin outside the ribs, and, worse still, into his lung, through allowing two years to elapse without reference to any doctor. This he did because near the beginning of the two years, two of the best-known continental specialists whom he happened to consult advised that the fluid be left alone.

I only mention such ancient history because I wish to emphasize that even to-day patients are not always so well advised and so lucky that no mishaps of this kind happen. Though there is an enormous gain in knowledge and experience of artificial pneumothorax, its complications and developments, there are still some gaps and shortcomings here and there. It is necessary to make sure that such

shortcomings are fewer and fewer, whether the patients are well-to-do or poor. More X-ray facilities, especially so that more frequent screenings can be done, are needed in many localities.

Ten years ago, and even more recently, I was much inclined to hold the view that if compression therapy seemed advisable, it was best in all cases first of all to try and induce a pneumothorax. If, and when, this was found impossible, other methods of collapsing the lung could be considered. To-day a man with the experience of E. J. O'Brien, of Detroit, says that he advises under many circumstances, resort to phrenicectomy before resort to artificial pneumothorax. He states (*American Journal of Roentgenology*, 1933, xxx, 318) "That it would be better to treat all patients with artificial pneumothorax if it were not that phrenicectomy is a simpler procedure and accomplishes the desired result in many cases. If, in any patient, the desired result is not accomplished promptly, artificial pneumothorax should be started." I know that a good deal of trouble is avoided if phrenic evulsion does what is required and that a career of artificial pneumothorax occasionally brings complications of obliterative pleurisy and hydro- or pyo-pneumothorax besides other worse complications. None the less, I am still inclined towards the view that artificial pneumothorax is probably preferable in the great majority of cases to other forms of collapse therapy. And it requires considerable experience and good judgment to pick out the minority for whom it is sound treatment to proceed straight to a phrenic operation. Again, under exceptional circumstances, it may be best to proceed straight away to thoracoplasty than to endeavour to get satisfactory results by any other method of compression, though I can hardly imagine any circumstances in which, if I were the patient, I should consent to a thoracoplasty before artificial pneumothorax had been tried.

The difficulty, however, is that some of these points are not yet ripe for final decision. It is only within the last five or six years that knowledge has advanced quickly in respect of such important matters as: (*a*) classification of cavities; (*b*) an appreciation of the importance of the presence of any cavity in the lung; (*c*) the practice of partial upper thoracoplasties; (*d*) the usefulness of phrenicectomy in a variety of different conditions; (*e*) whether phrenicectomy can be the cause of serious detriment in after years, and if so under what circumstances; (*f*) whether phrenicectomy always gives permanent protection to the patient and saves unnecessary restrictions in later life; (*g*) when and how to use oleothorax as a complement to artificial pneumothorax, a matter most assuredly requiring the close attention of an expert.

I am certain that modern treatment by collapse therapy has improved the prognosis considerably. This improvement depends upon three advances made in this century: (1) A more accurate knowledge has been acquired in recent years of the phases through which the disease in the lung passes. This is founded upon X-ray appearances, using screen and film appropriately. It presupposes skilled radiological technique, a good standard, and experienced interpretation of what radiology reveals; (2) an appreciation of what is happening to the patient. This assumes an intimate knowledge of the patient's state, and circumstances, his psyche and his economic conditions. However important the temperature, the pulse, the sputum, the digestion are, it is even more important, as a rule, to remember that it is the patient and not the disease which is to be treated, and that the patient is an individuum with a mind as well as a body. Aid from the physician in surmounting the all-too-frequent psychological handicaps which confront him may be more urgent than any physical treatment. This must perhaps be emphasized particularly this afternoon where our attention is being concentrated on what is, after all, a mechanistic form of treatment; (3) an up-to-date knowledge of the latest developments of collapse therapy.

One of the most experienced workers in America (E. T. O'Brien)[1] has said "The results of these procedures (of collapse therapy) will be almost in direct proportion to the intelligence with which they are applied." To illustrate what I mean let me give two brief quotations: (1) In the *Revue de la Tuberculose*, October 1933 (p. 843), A. Mauer and M. Kanony describe the good recovery of a woman aged 19 who had a right artificial pneumothorax induced in May 1931. In spite of the fact that some infiltration was present in the left upper lobe and despite the presence of adhesions towards the apex posteriorly on the right side preventing the compression of a big cavity in that region a good recovery was eventually made. Two thoracoscopic examinations showed that these adhesions could not be cauterized. So at the appropriate time (mark the last four words)—in May 1932—a resection posteriorly of ribs two to seven was undertaken. This led to the collapse of the big cavity and by continuing the then effective pneumothorax a gradual disappearance of symptoms was brought about. The second quotation is from a letter which I received a few days ago from Dr. Geoffrey Marshall:

"Could you admit in about ten days time, a Mrs. X, aged 33, who is at present in a private room at the Brompton Hospital under Tudor-Edwards? Her disease started in 1930, when X-rays showed tuberculous cavities in the right upper lobe. I induced a right A.P. and she did well for nearly two years, but then the disease commenced in the opposite lung. I then treated her with bilateral A.P., but the first lung re-expanded, with obliterative pleurisy and one of the cavities reopened. Tudor-Edwards has performed a very efficient right upper thoracoplasty, and the left A.P. is being continued. Mrs. X is in quite good condition, and is free from pyrexia and sputum. She is an exceptionally good patient psychologically, and despite all set-backs, I believe she will do well."

Mr. J. E. H. Roberts said there were one or two points he should like to advance.

On the first point Dr. Vere Pearson had anticipated him—i.e., the right time for carrying out these various surgical procedures. Artificial pneumothorax was at present—and rightly—entirely a question for the physician, but when it became a matter of further procedures, the surgeon was called in. He (the speaker) saw many cases in which he believed the physician was right in having suggested thoracoplasty but, he thought, not at the right time. He had a case in a girl, aged 19, for whom he felt that thoracoplasty was the right treatment, but as she had had no ambulant treatment, and he thought she might well be put to bed and watched until the proper time could be chosen for the operation, he had declined to operate at the time. He hoped to do so later.

From the surgeon's point of view, physicians could be divided into three categories in this respect: (1) Those who called in the aid of a surgeon to perform collapse operation only slightly anterior in time to the signing of the patient's death certificate. The number of these physicians was diminishing. (2) Those who believed that the surgeon could perform miracles, since they asked him to operate in cases which did need collapse, but who made their request at the wrong time. (3) Those who co-operated with the surgeon throughout the case, and, having concluded that some collapse would be necessary, decided with him as to the proper time and the particular form available for the patient in question. In many cases complete thoracoplasty was unnecessary; apicolysis was sufficient. If a previous phrenic operation had been performed, it was sometimes impossible to limit the operation in that way. Preliminary phrenic operation was often unnecessary in these cases, or, if carried out, should only be temporary, either by injection of alcohol or by compression of the nerve, so that it could later recover its function.

1 Loc. cit., p. 318.

Another point to which he wished to refer was the assessment of results. One constantly saw papers referring to results of thoracoplasty, and to results following avulsion, etc., but with no classification of patients, and with no mention of the indications in the particular patient or series of patients before any special procedure was adopted. It was impossible to get any valuable information from mass statistics of that kind. He had known cases in which the phrenic operation had been performed at the hospital because of pain in the diaphragmatic region, due to adhesions, and had immediately relieved the patient, yet such a patient might be dead in five years' time. Again, phrenic operations might be performed for urgent hæmorrhage. When one had taken a series of such cases and had allowed for the percentage who would have recovered in any case—as the hæmorrhage would have ceased spontaneously—there remained some in which it was legitimate to suppose that the phrenic operation had assisted in stopping the hæmorrhage. Those patients might die from the disease a few years later, but the operation was a success for the purpose for which it was performed. In the assessment of results there was now a better classification of patients and of indications for operation, and also a knowledge of what particular operation to perform. All thoracoplasties were not the same, and to classify on a merely limited operation was illogical.

Dr. James Watt said he was not sure that he had arrived at the same stage as Dr. Rist, i.e. of performing pneumothorax for minimal lesions: still, he was not so pessimistic as Dr. Wingfield as to the use of artificial pneumothorax in comparatively early cases. He thought that Dr. Wingfield over-estimated the dangers of pleural thickening and other occasional catastrophies. It did not seem fair to bring as an argument against pneumothorax or any form of collapse therapy an accident of rare occurrence, for instance a suppurating effusion.

Statistics ought to afford a good deal of information. It was incorrect to say that mass statistics relating to any particular kind of treatment were not of much value; it was only by comparing the results on large numbers of cases of similar nature that a broad opinion as to efficacy could be formed. He agreed with Mr. Roberts as to the desirability of forming subgroups, so as to compare results in one type of case with those in another.

In this discussion nothing had been said as to the results of phrenic evulsion in the final stage of treatment by artificial pneumothorax. It was felt that when a lung had healed to the stage when one thought of allowing it to re-expand, after three or four years of collapse, it was safer to reduce the volume of the thorax into which it could re-expand by doing phrenic evulsion. In many cases now a pneumothorax was finished off only after a preliminary phrenic evulsion.

The occurrence of effusions was the commonest bugbear in collapse therapy, and there was still much to be learned, not only as to the prevention of effusions, but as to the management of them. There was at present considerable difference of opinion as to whether these effusions should be left for a period, and if so, for how long they should be left. Some, including himself, thought the effusion should be drained off early, as it was safer not to allow the fluid to remain, on account of the dangers of adhesive pleurisy and the inevitable re-expansion of the lung before the cavities had healed.

Dr. L. S. T. Burrell: There are several methods of producing collapse or partial collapse of the lung, but I propose to deal chiefly with artificial pneumothorax, which should be regarded as a medical procedure. It is not permanent, for the lung can be allowed to re-expand by stopping the refills. Thoracoplasty, apicolysis, and phrenic

evulsion, on the other hand are surgical and produce a permanent effect upon the lung.

(1) *Artificial pneumothorax.*—Between 1919 and March 1923 I induced artificial pneumothorax in a series of patients with pulmonary tuberculosis. Most of them were referred to me because the condition was getting worse, and in every case simple medical treatment had been given for six months at least before pneumothorax was induced. None of them can be regarded as early or "good" cases.

I divided the series into three groups: (1) in which there was no evidence of active disease in the better lung; (2) in which there was slight activity in the better lung; (3) in which more than a third of the better lung was involved.

The following tables show the immediate and late results of artificial pneumothorax in these three groups. I have not included those patients who are alive but still have signs of active disease.

Group	Cases	Disease arrested		Dead	
		1924	1933	1924	1933
1	71	50	36	4	32
2	61	17	7	18	46
3	21	0	0	19	21

In a later series in which I induced the pneumothorax between March 1923 and November 1925 the results in December 1933 were as follows:—

Group	Cases	Disease arrested	Dead
1	61	35	24
2	61	13	38
3	24	2	21

If these two series are combined the results after at least eight years are as follows:—

Group	Cases	Disease arrested	Dead
1	132	71 (53·7%)	56 (42·4%)
2	122	20 (16·4%)	84 (68·8%)
3	45	2 (4·4%)	42 (93·3%)

From these cases it appears that, provided the disease is unilateral, over half the patients may be saved, even if the pneumothorax is not begun until six or more months' preliminary simple medical treatment has failed.

If, however, there is active disease in the better lung, only about a sixth can expect arrest of the disease, and if the better lung is extensively involved, pneumothorax offers no real hope at all.

In my opinion the lesson to be learnt from these results is that in unilateral cases with definitely active disease, pneumothorax should be induced early. It seems probable that if the pneumothorax had been started earlier in the cases in Group 1, a higher percentage than 53·7 would have been arrested, and if it had been begun before the better lung became involved in the Group 2 cases the results would have been much better.

The immediate results of pneumothorax are excellent and often dramatic, but the late results depend largely upon the stage of the disease when the treatment is started. There are certain cases of slight or so-called early pulmonary tuberculosis with a good chance of cure by simple treatment, and one must not forget that there are certain complications of pneumothorax which are serious, although it is true

that such complications are rare in the early unilateral case. I do occasionally see an early case with an artificial pneumothorax which has been induced, in my opinion, unnecessarily. For every such case, however, I see at least twenty in which I think the pneumothorax has been induced too late. When once the better lung becomes involved, the chances of an eventual cure by pneumothorax are enormously reduced, and if the better lung is extensively involved, pneumothorax is often more harmful than beneficial, although in some cases it does appear to have prolonged life.

As a clinician, I am frequently confronted with a patient who has recently become ill and who has definite, though slight, signs of pulmonary tuberculosis. Now the patient—and often his doctor—suggests that a few weeks on a farm or a sea voyage will effect a cure, and that he is not bad enough for sanatorium—still less for pneumothorax—treatment. For many years I have asserted, and I now repeat, that the duty of the doctor is to prevent a patient from becoming "bad." In my opinion it is wrong to adopt the attitude that it is justifiable to acquiesce in a patient's drifting unguided during the early stages of his disease because modern methods of treatment may, even in an advanced state, result in a cure. The good physician is the one who prevents his patient from getting into a dangerous position, and not the one who by some drastic method rescues his patient from a dangerous position in which, under more skilful management, he would not have found himself.

It often happens that when the patient is first seen there is already a considerable amount of disease in the lung. In this case pneumothorax may check the rate of spread and may stimulate fibrosis, but thoracoplasty will probably become necessary later to control the symptoms and complications of fibroid lung.

If there is active disease in the better lung, a bilateral pneumothorax may prolong life considerably, but the ultimate prognosis is always bad in these cases.

When the diseased part of the lung cannot be collapsed, owing to adherent pleura, it is usually better to try some other method of collapse, and not to persist in maintaining a partial collapse of the lung. A good thoracoplasty is better than a bad pneumothorax, but one must not forget that a bad pneumothorax may sometimes be converted into a good one by cauterization of adhesions.

(2) *Thoracoplasty.*—In my experience, this operation is of great value in a limited number of cases, and frequently saves the life of a patient who, without it, has no expectation but of gradual decline. It is not indicated in the early or mild cases, and the aim of the physician dealing with an early case should be to prevent his patient from getting into a state when thoracoplasty is necessary.

In spite of the best management, however, it does happen that a patient develops unilateral fibroid disease with activity, and in thoracoplasty one has a method of treatment which offers a genuine chance of recovery.

(3) *Apicolysis.*—This method of treatment is, in my opinion, the best for dealing with an apical cavity or local apical disease when the affected part of the lung cannot be collapsed by pneumothorax.

(4) *Phrenic evulsion.*—As a means of treating pulmonary tuberculosis this operation has been found wanting. In certain cases, e.g. as a preliminary to apical thoracoplasty, it is actually harmful. As an adjunct to pneumothorax, or to ease the condition when the mediastinum is displaced, or as an aid in a complete pneumothorax, it has its value.

Dr. Frederick Heaf: After hearing the convincing statements of Dr. Rist and Dr. Burrell, no one will doubt that the results of artificial pneumothorax treatment are good. On the other hand, everybody will agree that in certain artificial pneumothorax cases the results are not successful. This may be for two

reasons : (1) The patients may have no resistance to the infection. (2) The lung cannot be collapsed owing to adhesions. In cases having no resistance to infection it is doubtful if any form of treatment will be of use. In the second the failure of the artificial pneumothorax treatment is due to adhesions which keep the lung under tension and prevent its relaxing. A lung under stresses and strains of tension due to adhesions rarely heals completely. We therefore must release the lung by some means so that it may relax. When the adhesions can be cut, I have found that there is little danger from pleural effusion if a galvano-cautery is used instead of diathermy. If the adhesions cannot be cut, then phrenic evulsion or thoracoplasty is definitely indicated. This argument of the harmful effects of apical adhesions applies to early untreated cases, and I have found marked contraction and occasional obliteration of cavities after phrenectomy in such cases. The operation releases the tension on the lung tissues.

In supporting the remarks of Mr. Roberts, I wish to emphasize our need for a standard of guidance in treatment and comparison of results. Temperature, pulse-rate, X-ray examinations are at times all unreliable. My colleague, Dr. Houghton, has given me permission to speak of the work he is doing on the value of blood-counts in assessing progress. He finds that often blood examination shows no improvement in cases which have apparently done very well on routine sanatorium treatment, whilst a marked improvement is often shown in cases to which collapse therapy has been applied.

Finally, I should also like to stress the value of rest combined with all forms of collapse therapy. The enforcement of a period of absolute rest before beginning any form of collapse treatment will greatly add to the ultimate success of the treatment in all cases.

Dr. F. G. Chandler : I should like to ask Dr. Rist what he means by " cure " in bronchiectasis ? Does cure persist after the termination of the artificial pneumothorax, or does he continue with the artificial pneumothorax throughout the patient's life ?

With regard to artificial pneumothorax in general, increasing experience strengthens my belief that it is one of the most successful methods of treatment ever devised. Concerning its indication there is not universal agreement. I cannot subscribe to the view that the treatment should be applied to practically every early case ; I would give nature a good chance. To my mind, the indication—in the early type of case—is a lesion spreading or breaking down, or failing to heal, in spite of treatment on sanatorium lines, irrespective of the size of the lesion.

Probably most of us will agree that the early promise of oleothorax has not been fulfilled. It has disappointed and even frightened us. Nevertheless, I think it still has its place. It is the only way of maintaining collapse if obliterative pleurisy sets in. I will quote one case : A young man was admitted to hospital under my care, in 1925. He was desperately ill with severe diabetes and a galloping broncho-pneumonic phthisis in the left lower lobe. Artificial pneumothorax, diet, and 60 units of insulin a day restored him to health. A large effusion developed, however, which was tapped, recurred, and was tapped again. Obliterative pleurisy set in. Something had to be done, and so a large oleothorax was produced. For six years he kept in good health, and at full work, except for two attacks of mild diabetic coma. In 1931 he developed coma again and died. I obtained the lungs for examination. The tuberculous lesions which were extensive, acute, and severe, had completely healed. The pleura was so thickened that I cannot believe that the oil could ever have ruptured through into the lung, which is one of the dangers feared. In fact I believe that this accident will happen only when there is active tuberculosis

persisting in the lung. If the disease, therefore, remains uncontrolled by the oleothorax, this surely is one of the indications for its termination.

Dr. Rist (in reply) said he agreed with Mr. J. E. Roberts as to the difficulty of forming a judgment from statistics. Dr. Wingfield had said that the cases should be classified, but there was no classification of the various forms of tuberculosis which satisfied everybody; each physician had his own. Nor did he think one could judge by giving collapse therapy or pneumothorax to one set of patients, and to others some other form of treatment. He (Dr. Rist) did not think that could be done in the disease under discussion: if one believed in pneumothorax, it was unfair to neglect to give every patient the benefit of it just for experiment. But one might be saved from the responsibility of an arbitrary choice by the patients themselves, as he (the speaker) had shown in statistics first published in Washington in 1926, and since then in extended form in France.

Patients (given approval as to fitness for the operation by the physician) were divisible into two classes: (1) those who accepted pneumothorax, and (2) those who refused it. Whatever form of tuberculosis they had, he thought the two classes could fairly be compared. In the figures referred to the outlook of the patients who had refused pneumothorax was significant; that was to say, after a few years a percentage of only four or five remained alive. They were not healed, and were in a bad condition.

On the other hand, the pneumothorax cases had a survival percentage of about 50%, and among these about 35% were able to work, having been permanently restored to normal life.

He (the speaker) completely agreed with Dr. Vere Pearson as to the necessity of seeing in the patient not only a "case," but an individual, and not only an individual but a member of a household, a man who had a profession, responsibilities and ideals, and whose psychological as well as economic conditions should be taken into account. It was as a rule necessary that such a man should be restored to his work with as little delay as possible. Those who were so fortunately placed that they could afford to lead almost indefinitely the life of an invalid, and spend long periods year after year in a sanatorium, formed a continually diminishing proportion of the total number of patients. The others, the constantly growing majority of economically dependent patients, constituted a strong argument in favour of early pneumothorax. He had met many young patients, such as students, who did well with sanatorium treatment, but the effect was utterly lost after two or three years because they relapsed. This relapse was due to the fact that they could not be kept at perfect rest—their economic circumstances would not permit it. It was now his custom to carry out collapse in such cases as soon as possible, and it was his view that he thereby saved the patients from years of invalidism, and restored them earlier to complete health.

He was less anxious about the danger of effusions than Dr. Wingfield was. Not only did these effusions often disappear without doing damage, but even when they caused pyopneumothorax and a pleural cutaneous fistula developed, the patients might recover perfectly. Some of his older patients were still alive and working, and their recovery had lasted eighteen, twenty, and twenty-two years. Some of them had had pyopneumothorax for some time, and they also had fistula, and they had been at work for many years.

There were even some kinds of effusion which were entirely beneficial to the patient. No one who had had long experience could have failed to notice such cases, in which pneumothorax collapse had given a tolerably satisfactory result, the general symptoms having disappeared, but the collapse being not altogether complete: a small cavity remained open, there was still some expectoration, and bacilli could be

found in it. Suddenly an effusion took place, with all its disagreeable symptoms—pain, fever, loss of weight. And coincidently with the appearance of the effusion the bacilli disappeared from the sputum. After the effusion had become absorbed and the patient had recovered from it, the progress towards complete recovery was steady and rapid. Such cases were observed right at the beginning of the pneumothorax era. Von Muralt mentioned them as early as 1913, and thought they could only be explained immunologically by what he called the serum action (Serum-Wirkung) of the fluid. Experiments made by himself (Dr. Rist) with Weber and Jonesco gave some support to that explanation. There were fluids occurring in a pneumothorax which exerted a decided bactericidal action on tubercle bacilli.

Section of Urology

President—A. CLIFFORD MORSON, O.B.E., F.R.C.S.

[*November* 29, 1933]

Renal Tuberculosis.—CUTHBERT DUKES, O.B.E., M.D.

It is well known that in some cases of renal tuberculosis, tubercle bacilli are easily found in the urine and in other cases they are scarce or absent. Whether tubercle bacilli are few or many does not depend directly on the stage of the disease but depends on the situation of the tuberculous lesion within the kidney and the condition of the renal pelvis. This is illustrated in the ten specimens exhibited, all removed by nephrectomy for unilateral tuberculosis. Urine examination before operation had revealed tubercle bacilli in only six of these cases. When these are compared with the cases in which tubercle bacilli were not found, it will be seen that in all the positive cases there is a distinct communication between a tuberculous cavity and the renal pelvis, or else an ulceration of the renal papillæ or a tuberculous pyelitis.

The specimens in which tubercle bacilli were not found in the urine are of two types. In one the renal pelvis is normal, the tuberculous lesion being embedded in the kidney and shut off from the calyces by a zone of healthy tissue. In the other the renal pelvis is obliterated by fibrosis, or a stricture cuts off the communication between the kidney and ureter. One of the kidneys exhibited—from a case under the care of Mr. Alban Andrews—has a double ureter and a double renal pelvis (see fig., p. 10). Before operation each ureter was catheterized and tubercle bacilli were found in the urine from the upper renal pelvis, in which an ulcerating tuberculous lesion is visible. No tubercle bacilli were found in the urine from the lower ureter though the portion of kidney drained by this renal pelvis is also tuberculous. The difference is that in this region the lesion consists of a cavity containing viscous material but this cavity does not communicate with the renal pelvis.

This distinction between open and closed tuberculous lesions is, of course, well known, but I thought it would be of interest to show a series of specimens illustrating it, and to describe how it may be demonstrated. A good plan is to distend the renal pelvis with formalin and then ligature the ureter and leave the specimen to fix in 10% formol saline for from two to three days. The dissection is carried out by first exploring the renal pelvis through a V-shaped incision in the anterior wall, then slicing the kidney with a long sharp knife exactly through the

middle, and finally opening up each calyx with a small pair of scissors. An early tuberculous ulcer at the apex of a papilla may easily be overlooked unless the kidney is examined in this way.

I should like to take this opportunity of referring to the value of cultures for tubercle bacilli. Recent improvements in bacteriological technique have made it comparatively easy to cultivate the tubercle bacillus, and when acid-fast bacilli resembling tubercle bacilli are found in the urine, the characters of the culture growth can be made use of for identification. A good growth of tubercle bacilli is generally to be obtained in three or four weeks. When no tubercle bacilli have been seen in

Tuberculous kidney with double renal pelvis and double ureter. Tubercle bacilli were found in the urine from the upper renal pelvis, in which there is an open tuberculous lesion. No tubercle bacilli were found in the urine from the lower renal pelvis although this drained an area of the kidney embedded in which is a caseating tuberculous lesion.

stained films from tuberculous urine, the cultures are positive in almost the same proportion of cases as are shown to be positive by guinea-pig injection. These views are based on 160 cases in which I have used the culture method for diagnosis. Confirmation of the presence of tubercle bacilli was obtained by culture in almost every case in which the stained films had been positive. In one case, and one only, in this series the cultures were positive when direct films were negative, and in this case the guinea-pig test was also positive. Compared with the guinea-pig method, the culture method of diagnosis is much quicker and cheaper, and avoids experiments on animals.

A Large Ureteric Calculus passed Spontaneously into the Bladder.—H. P. WINSBURY-WHITE, F.R.C.S.

The patient was a woman, aged 28, who was suffering from bilateral urinary calculus. There was a large branched stone in the left kidney and an elongated stone about 1½ in. in length in the right ureter just above the pelvic brim. After cystoscoping this patient and inspecting the skiagram I decided to admit her to hospital for open operation for the purpose of removing the stone from the right ureter. Judge of my surprise when the skiagram taken a week later, just after admission to hospital, showed that the ureteric stone was then in the bladder. Thus I was able to remove the stone by litholapaxy instead of by an extensive open operation.

There are two points of special interest in this case: one is that a stone of such a size could find its way spontaneously from the ureter into the bladder, the other is that the passage of the stone should take place at the time that it did. No trans-cystoscopic manipulations of any kind were carried out on the ureter during the cystoscopy, but there is no doubt that the distension of the bladder with fluid will often set up the necessary activity of the ureter which induces it to try to expel a stone which may be present.

A year or so ago I had a somewhat similar experience with a stone in the lumbar portion of the left ureter in a man aged 38, who also had stones in the right kidney. The ureteric calculus was just under half an inch in diameter and rounded. Here again, soon after the cystoscopy I admitted the patient to hospital for open operation for removal of the stone in the left ureter. The skiagram taken while the patient was in hospital showed the stone in the bladder. This I was also able to remove by litholapaxy. I have noticed the same thing happen in a number of cases where smaller stones have been present in the ureters, but these two cases are outstanding, having regard to the dimensions of the calculi in question.

Calcification of Pelvic and Lumbar Glands in Chronic Prostatitis.—H. P. WINSBURY-WHITE, F.R.C.S.

The patient was a man aged 73, and the skiagram shows well-developed prostatic calculi, and, in addition, calcified areas marking out a chain of glands on either side of the hollow of the sacrum. There is also a large calcified gland in the lower lumbar region of the right side, between the tips of the transverse processes of the fourth and fifth lumbar vertebræ.

I am showing this lantern slide because it is an outstanding example of the fact that chronic prostatitis commonly gives rise to calcification in the lymphatic glands along the route mentioned, so that when these features are seen in cases in which prostatitis is not suspected, interest should at once be directed towards the condition of the prostate.

Hypogonadism.—E. W. RICHES, M.C., M.S.

Male, aged 27, one of a family of six, of whom the others are normal.

Previous history.—Nothing relevant. He has never had mumps.

History.—At the age of 18 he became aware that he was different from his fellows. At the age of 22 his voice began to break, and pubic hair appeared. His

Hypogonadism: General appearance.

Hypogonadism: Facies.

Hypogonadism: External genitalia.

height increased from 4 ft. 9 in. at the age of 22, to 5 ft. 6 in. at that of 27. He has occasional erections but no emission.

Present condition.—Spare build, infantile facies, voice high-pitched. No hair on chin or in axillæ; scanty pubic hair.

Penis 2 ins. long; testes each the size of a pea.

Pituitary fossa normal radiographically; visual fields normal.

Most of the epiphyses are un-united.

Subsequent History of a Case after Per-Urethral Prostatic Resection.—A. RALPH THOMPSON, F.R.C.S.

R. H., aged 64, was admitted to Guy's Hospital under the care of Dr. E. P. Poulton, on November 3, 1933, suffering from moderately severe hæmaturia, with a marked degree of glycosuria. In 1926 a stone was removed from the left kidney, and in 1928 a papilloma was destroyed by diathermy through a cystoscope.

In 1932 the patient was subjected to per-urethral section of the prostate gland at another hospital. The main symptom for which this operation was performed was frequency of micturition. After the operation he was much better, and comfortable till about a week before he was admitted to Guy's Hospital, very ill and obviously dying.

Mr. E. H. Rink opened the bladder by the suprapubic route. It was found to be of very small capacity, and filled with a large mass which at first appeared to be malignant. A drainage tube was inserted into the bladder. Two days later the mass separated, and was voided through the suprapubic opening.

The mass was solid and firm to the touch, and measured 4 in. long by 2 in. broad by 1 in. thick. On section it was seen to be composed of mucous tissue.

The patient died twelve days after admission.

Post-mortem examination showed acute hæmorrhagic cystitis, and the bladder wall infiltrated with growth, especially on the left side. The prostate appeared normal. There was bilateral pyelonephritis, old on the left side, recent on the right. The left ureter and the pelvis of the left kidney were enormously dilated.

Mr. ALEX. E. ROCHE, F.R.C.S., showed a case of **Silent Renal Tuberculosis.**

Section of Psychiatry

President—E. MAPOTHER, M.D.

[*January* 9, 1934]

The Medical Aspects of Crime

By W. NORWOOD EAST, M.D., M.R.C.P.

(*H.M. Commissioner of Prisons, England and Wales*)

THE prevention of crime and the provision of alternatives to imprisonment in suitable cases are important problems of social hygiene, and medical questions are frequently involved in their solution. In connexion therewith it is important to bear in mind that conduct which outrages the moral sanctions of society is not necessarily criminal in the technical sense, and that criminal conduct may result from altruistic motives in normal as well as abnormal persons. The scientific approach to crime must consider all those acts of omission or commission which render the subject amenable to the criminal law, and the medical aspects of crime may be limited accordingly.

I do not propose to consider the profound and fundamental problems of heredity and freewill, although they are of constant interest in the study of criminal conduct. The influence of the former in any individual case may be inferred, as we know, only after long, careful and exacting research. In regard to the latter, I suggest that the academic disputes between determinists and their opponents have failed so far to assist materially the practical problems associated with crime.

Broadly speaking, the medical aspects of crime can be divided into the physical and the mental, and the sometime association of permanent or temporary physical or mental disease or disability with crime is recognized generally. It is often obvious. But in my opinion statistics of adult crime would be little affected if all the gross and minor physical defects, septic foci, endocrine disorders and the like were relieved at vast public expense.

The medical aspects of criminal conduct are concerned primarily with individuals, the legal point of view with the protection of society, and it is well to remember that the value of medical opinion in the criminal courts would soon become negligible if this protection was withdrawn.

The medical outlook on crime has suffered in the past from the statement that crime is a disease. It is difficult to accept this generalization in our present state of knowledge, particularly if the large number of people is considered who commit crime as the result of unpopular motor or other legislation. Like many over-statements, it has diverted attention from the real facts, and here, from the fact that both social and anti-social conduct present mental problems which are recognized now to be often more complex than was believed formerly.

The scientific approach to crime is not limited to the investigation of pathological states of mind and body, for the best method of attack for practical purposes may

be sociological, educational, or penal, and not medical. It may be unnecessary therefore to decide always the precise degree of abnormality associated with any particular crime. It is occasionally harmful to do so, for the delinquent should not be encouraged to believe that he is an irresponsible mental invalid. Further, every individual who reacts to similar circumstances in a different manner from that in which his fellows do, does not necessarily require medical attention, since a wide range of reaction to various stimuli is sanctioned by society. Havelock Ellis has recently emphasized the fact that in psychic health, to an even greater extent than in physical health, the range of what may be considered normal variation is very wide. And further, he states in regard to sexual reactions, that, since we are all made up of various impulses, the sexually normal man is often a man who holds in control some abnormal impulse [1].

It is a matter of common knowledge that the motives, intentions, and temptations which cause crime in certain individuals may be inoperative in others, and the suggestion is made sometimes that if a crime is the result of slight provocation the fact may be regarded as an indication of mental abnormality: as when a woman steals from a store an article she can well afford to purchase. Usually, however, all the facts connected with a crime, as well as the past history and present condition of the offender, must be considered before conduct can be regarded as abnormal and outside the limits of the ordinary variations of persons of the same class and upbringing when subjected to similar circumstances or temptations. An incorrect diagnosis may be avoided only by familiarity with these reactions: for example, with the large amount of shoplifting practised by well-to-do women. Indeed, experience in the criminal courts proves that the judicial authorities, and others officially concerned with criminals and criminal conduct, usually arrive at reliable conclusions in regard to the motives and intentions of law-breakers.

In doubtful cases the frequency of the crime, and personal bias—including the tendency to consider one's own standard of conduct as normal—may affect opinion. Homosexuality, for example, may be regarded as normal or abnormal, but whether homosexual conduct is due to congenital inversion, premature arrest in sexual evolution, acquired sexual deviation, or commercial cupidity, does not affect the point I wish to make, namely, that anti-social conduct appears to be less certainly attributable to abnormality of mind in proportion to the frequency with which the corresponding anti-social tendencies are found in the ordinary population, and the degree in which they are present in the assessors.

I have used the term anti-social conduct. I should add that when applied to criminal conduct it is acceptable usually in a limited sense only, for the criminal may be anti-social in one direction or in many. Even in the latter case he usually shows gregariousness towards a smaller herd composed of types similar to his own. This may be seen in any prison where communal behaviour is exploited by the administration for the employment of prisoners at co-operative labour; conversely, it may lead to attempts to overthrow authority. Perhaps the most complete negation of the herd instinct is found in certain cases of mental disorder and mental defect, but striking examples are met with also in some criminals who cannot be included in these categories.

The bald statement is made sometimes that the punishment must fit the criminal; but the punishment must fit the crime also. If anti-social conduct is associated with mental disease, illness or defect, it can be dealt with in a manner acceptable to legal, medical, and public opinion. If it results from a faulty personality which is outside the scope of the Lunacy, Mental Deficiency, or Mental Treatment Acts, the above dictum in certain cases would involve permanent deprivation of liberty for an offence which was not in fact a social danger. The effect might be to provoke serious crime in order to avoid the penalty for a nuisance. Moreover, public opinion, the final arbiter, does not appear to be prepared to sanction

prolonged detention for a nuisance, however favourable the custodial conditions may be.

On the other hand, if a crime result from deliberate and malicious intent, the public will demand protection from the determined aggressive criminal and his copyists, and if dissatisfied with the protection afforded by authority, will endeavour to satisfy its desire for social safety by other means. There seems to be little doubt that lynch-law is prompted often by the desire for revenge, because the self-regarding sentiment is outraged and insulted. But circumstances arise from time to time among people not given to this method of self-expression, which binds them together by fear of some repeated criminal act in their locality and leads them to adopt measures of safety independent of police action.

Crime is essentially a social problem, and not altogether a medical one. To ensure permanent advantage from medical methods of attack, the approval of legal, medical, and public opinion is necessary. This is of the first importance, but is sometimes forgotten, and we have seen in our own time medical efforts fail when this support has been withheld or withdrawn.

It is common knowledge that dissatisfaction is felt in certain quarters with regard to the legal restrictions in cases of alleged criminal irresponsibility, and the discussions on the subject are well known. Can there be any doubt that the criteria of criminal responsibility remain unchanged because they are accepted generally and the suggested amendments fail to convert the main body of opinion?

It will be remembered that during the Great War, Regulation 40 D of the Defence of the Realm Regulations made it a summary offence for any woman suffering from venereal disease in a communicable form to have or solicit sexual relations with any member of His Majesty's Forces. If this emergency measure had been modified to apply to peace conditions and received general support after the Armistice, the prevention of venereal infection by means of penalties might have continued.

The history of the Inebriates Acts is of interest in this connection. The Habitual Drunkards Act, 1879, merely enabled non-criminal inebriates to submit themselves to voluntary control. After this Act had been in force for some years public attention was again directed to the problem of inebriates who refused control and rendered the Act largely ineffective. The Departmental Committee of 1893 followed, and its recommendations led to the Inebriates Act, 1898, which related to criminal inebriates. In consequence two State Inebriate Reformatories and thirteen certified Inebriate Reformatories were established, and the Act received at first a considerable measure of legal, medical, and public support, but this was ultimately withdrawn. The State Inebriate Reformatories were closed during the Great War, and the last certified Inebriate Reformatory in 1921. The failure of the reformatories was attributed to defects in the Act itself, the reluctance of Committing Authorities to send suitable cases for cure, the refusal of some authorities to continue payment, and the result of experience which showed that compulsory deprivation of drink was in itself no cure for drunkenness. Perhaps no cure was possible, for the addiction was firmly established before the inebriate came within the sanctions of the Act, and modern methods of treatment were not generally applied. In this connection it may be recalled that the Departmental Committee on Persistent Offenders in 1932 was confronted with opposing medical views in regard to the treatment of inebriety [2]. Some witnesses considered that psychological methods were frequently beneficial, others found the results of this form of treatment were discouraging. However this may be, the Inebriates Act of 1898 failed, in spite of the fact that it was the outcome of expert opinion. It was, nevertheless, an important and necessary medical and social experiment. It is to be noted that the subsequent legal and social experiment, the restriction of the hours during which drink can be obtained, has met with remarkable and continued success.

The association of legal, medical, and public opinion has had a happier result in

more recent times in connection with the Mental Deficiency Acts, and the public support the compulsory detention of defectives who have committed crime. This is due largely to the fact that the medical principles relating to mental deficiency are held to be satisfactory, but if the occasional observations of judicial authorities are studied, there can be little doubt that legal and public support will be withdrawn and the influence of the Acts on criminal problems be forfeited if extreme medical views concerning the relation of crime and mental defectiveness become general.

I make no excuse for drawing attention to these well-known facts, because they are fundamental. In spite of this they are frequently disregarded in discussions concerning the scientific treatment of crime. No progress can be expected in this direction unless the judicial and administrative authorities and the public are satisfied that modern therapy can be relied upon to produce beneficial results in certain cases of crime.

Closely allied to this matter is the fact that recent advances in general medicine, although great in many directions, have been less effective in others. Perhaps this contrast tends to exaggerate sometimes the benefits to be derived from new methods of treatment. Also T. A. Ross [3] has told us that psychotherapeutic methods used successfully by him at first, failed to produce good results as soon as he came to doubt the efficacy of the treatment, and this experience seems to be not uncommon. But if new therapeutic measures fail to fulfil their early promise, their true scope and value may become distorted. Only experience can demonstrate their limitations as well as their superiority. If, too, a ubiquitous press takes a hand in the matter and arouses public interest, the early enthusiasm may be replaced by unwarranted adverse criticism and mistrust if striking failures occur.

It is suggested that expert opinion to-day does not always command unquestioning confidence. The public, viewing the frequent conflicts of opinion among experts in general, in the Courts, in debating societies, and elsewhere, has come to doubt their infallibility and is left to wonder whether acutely antagonistic views can be harmonized with actual facts. Looking, too, at the world-chaos to-day, and finding little relief from the remedies advanced by opposing schools of thought, it turns aside and demands proof, and is intolerant of unsupported speculations and pontifical assurances.

The psychological treatment of delinquency and crime is to all intents and purposes a post-war growth as far as this country is concerned, and perhaps no medical psychologist in this country is greatly experienced, so far, in the matter. In spite of this we are told sometimes that the present legal means of dealing with crime should be replaced by psychological methods of dealing with criminals, regardless of our ignorance and the many practical difficulties involved.

Recently G. Pailthorpe [4] has outlined a system which it is suggested could be started as an experiment, and the details of its working carried out in accordance with the premise that crime is a symptom of underlying defect or disease. This system is to be divorced entirely from the present penal system which it is to replace apparently at some future date. The offenders are to be studied in buildings which are to be called hospitals, and are to be sent in for investigation at the time of their first offence. They are to be handed over in small groups to an investigator representing one or other of the several different schools of psychology, and are to come under his sole guardianship. We are informed that the psychological investigator may prefer to conduct the experimental treatment of the subject in his normal environment, or make some change in that environment although allowing the subject to carry out his normal occupation at the same time. It would appear that anyone who commits an offence which is punishable by a fine of a few shillings may be handed over, under this system, to a psychologist who might, or might not, permit him to carry out his own business or profession whilst undergoing a prolonged systematic psychological investigation. Further, it would appear that the psychologist might allow the rapist, the fire-raiser, the fraudulent company promoter, the burglar,

the murderer and other criminals, freedom and facilities to commit further crime. The proposal, in short, and as far as I understand it, would give the psychologist power to detain a delinquent whose offence might be dealt with properly under the Probation Act, and set at liberty the dangerous criminal from whom the public require protection!

Stress is laid on the fact that the place of detention in which the psychological investigation will be carried out is to be called a hospital and not a prison, but whatever name serves the institution, it becomes a prison as soon as detention therein is compulsory. Importance is attached also to the fact that because the institution is called a hospital and not a prison, no stigma will be felt by the residents. But is it desirable to remove altogether the stigma when this is a factor in the prevention of crime? Can anyone believe that it is desirable to suggest to law-breakers that they are mental invalids? Can anyone doubt that society and the criminal are better served if the latter can be made to realize the necessity to cultivate a sense of social responsibility?

We are not told how the offender is to be dealt with who refuses to co-operate with the psychologist, and there will be many. Also, we are left to imagine the indignation of a Watsonian delinquent when subjected to hours of compulsory association with a Freudian investigator.

It is an essential element of British justice that an accused person is considered to be innocent until the contrary is proved. It is assumed sometimes, however, that as soon as a person is accused of an offence he becomes a fitting subject for an intensive mental analysis. Too often no distinction is made between the status of the law-breaker before and after conviction. Too seldom is it realized that criminals in general, unless they are malingerers, resent the suggestion that their mental condition is a fitting subject for inquiry.

An accused person is allowed free consultation with his accredited legal advisers, and, if in custody, out of the hearing of officials, in preparation for his defence. Can we believe that he will pay more attention to the medical psychologist who is trying to unfold the antecedents to the crime, or to his legal adviser who is building up his defence against the charge? The two points of view will be often opposed.

Another element of British justice is the celerity with which trial and sentence follow on the commission of a crime. Imprisonment may be delayed temporarily if the prisoner is given leave to appeal against the conviction or sentence; it might be delayed for many months if it was not implemented until the result of a prolonged mental analysis was known. It is difficult to believe that the anxiety of the convicted prisoner, consequent upon this delay, would be helpful to the mental investigator. That it would be detrimental to the mental health of many offenders is certain. Further, the delay would serve no useful purpose if the analysis was negative or inconclusive.

The administration of criminal justice in this country has been built up through the centuries. The Criminal Law of Ancient Rome is acknowledged to have exercised in many ways an influence on our own law. It is unnecessary here to consider the origin of, and introduction into, criminal justice of the Assize Courts, the Central Criminal Court, the County and Borough Courts of Quarter Sessions, the Courts of Summary Jurisdiction, and in our own time the Court of Criminal Appeal. It is generally recognized that there is no country where the law is administered with such reliability and exactness as in England. Moreover, the criminal law is not static; changes are effected from time to time as occasion demands and the vast experience of highly skilled legalists directs.

Little progress in regard to the scientific treatment of crime can be expected as long as there is any considerable body of opinion which accepts the view that the law confuses sin and immorality with crime, and that sentences are imposed and carried out in a spirit of vindictiveness and hate. It is idle to ignore the facts.

Juries are constantly informed by the judiciary that they are not concerned with the moral aspect of the case, but only with the guilt or innocence of the accused; judicial authorities from time to time postpone sentence in order to review the evidence free from emotion, if revolted by the nature of the crime and the callousness of the criminal. Courts of Appeal function and reduce sentences which are regarded as inappropriate; prisoners frequently admit that the sentence imposed upon them for a serious crime is considerably less than they expected to receive; sentences are not carried out in an atmosphere of hate and revenge; prison officials are not less humane than ordinary citizens; prisoners are constantly visited by members of the Visiting Committee, to whom they may complain; they frequently see the prison visitors and their own friends; prisons are frequently visited and inspected by the Commissioners of Prisons and the Assistant Commissioners, who mix freely with their charges; the Secretary of State may be approached in writing by any prisoner. No one with any knowledge of the subject could persuade himself honestly that prison administration in this country to-day is harsh or revengeful. Its aim is the reformation of the law-breaker and his readjustment in civil life.

It is suggested sometimes that since the cure of the offender lies in the hands of the psychotherapist, his discharge to liberty should be also under the same direction. Can we believe for a moment that public opinion would accept this? A physical disease may be considered cured if the subsequent history and re-examination of the patient are negative. Similarly with mental disorder, but here the after-history is all important, and the inquirer may be misled purposely in his reinvestigation by the interested patient and his friends or accomplices. Unless the investigator can check the subsequent history of a criminal case from external sources, he is liable to estimate incorrectly the result of treatment.

It is an unfortunate fact that the public are confidently informed sometimes that a law-breaker requires medical treatment and not imprisonment, although the author of the statement has not examined the delinquent and has no information concerning the circumstances of the offence, except such as may be derived from the columns of the sensational press. To criticize, under these conditions, legal decisions and the conclusions of medical experts who have examined the offender and considered the facts connected with the crime can serve no useful purpose. A sentence of imprisonment imposed upon a man who repeatedly cut off girl's plaits of hair was said to be barbarous, and it was alleged that the offender was clearly in urgent need of medical treatment. The critic was unaware that the offender sold the plaits for five pounds each and had no sexual interest in them. But the magistrate who tried him and the medical expert who examined him were acquainted with the prisoner's frank admissions concerning his commercial undertakings.

It would be incorrect to believe that the mental condition of an offender is only taken into consideration by the courts in cases of insanity, as mental defectiveness is a condition which is considered almost daily by the judicial authorities when dealing with law-breakers. Moreover, the criminal courts frequently deal with the offender who is not insane or mentally defective in accordance with the medical evidence. I have been asked to advise the Judge at Assize four times in one afternoon in regard to prisoners who were not insane or mentally defective, and in each case my suggestions were adopted, but clearly the judge and not the doctor must decide how the public interest will be served best. A medical psychologist, when giving evidence before the Departmental Committee on Persistent Offenders, put the matter thus: "Psychologists tend to ignore the major social function of the penal system, but they should be reminded that the community is entitled to the first claim, and the individual only to the second. The business of the psychologist is merely to point out methods that are just and that may be fruitful in the individual case, provided that such methods are compatible with the wider interests of society" [5].

It has been alleged that no homosexual has ever been cured by jail. It is true that if the homosexual offender debauches boys, he will probably sooner or later achieve notoriety and the attentions of the police. It is true also that if he selects as his companion an adult homosexual, their practices may remain secret. My own experience, however, leads me to believe that a sentence of imprisonment does prevent at least some homosexuals from further delinquency. It is difficult to believe otherwise if the offender is attracted to boys only and does not return to prison.

There can be no doubt that imprisonment often fails to check sexual deviations, but it is to be remembered that some sexual offenders refuse psychological treatment lest it cure them of their anti-social pleasures. In ordinary life various sexual irregularities are not infrequent, and the sexual impulses of inverts and perverts, like those of normal individuals, vary widely in intensity. In some the sexual urge is controlled with ease, in some with difficulty; in some it is uncontrolled. It would be contrary to experience to believe that imprisonment is ineffective when the impulse is weak, or that it may be never effective if the impulse is strong.

Perhaps the psychotherapist is not the best person to determine the matter, for the patients who apply to him for treatment will be those who have most difficulty in controlling their desires. Also the literature on the subject is impressive mainly because the cases recorded are extreme instances of the condition. It is instructive to remember that an invert has written: "There are many inverts who, despite strong temptations, lead completely continent lives, and others who, like perfectly normal men, suffer few temptations of the flesh. . . . The class of inverts whose cases are most frequently studied are not typical examples of the silent and generally decent thousands whose burden is a temptation rather than a sin" [6].

It is suggested sometimes that the psychological approach to crime should be parallel to the legal approach. It is clear that an intensive and sometimes prolonged investigation by the police into the circumstances connected with a crime may necessarily precede an arrest, and that there can be no parallelism at this stage.

After arrest and before trial there is the very real difficulty that a mental analysis may disclose important information concerning the crime, and that a medical man is not empowered to withhold the information from the police authorities in charge of the case. Neither can he claim the privilege of professional secrecy in the witness box. Here again there can be no parallel.

It will be remembered that Section 8 of the Mental Deficiency Act, 1913, provides a court of competent jurisdiction with certain alternatives to imprisonment or penal servitude in the cases of defectives who are convicted of crime. It is to be noted particularly that the sanctions do not become operative until the accused is found guilty. In a somewhat similar manner the psychological and legal approach to crime may run parallel in certain cases, for a psychological investigation may also suggest to the judicial authority alternatives to imprisonment or penal servitude. Nevertheless it may be considered proper to impose the legal consequences for a crime although it is due to an abnormal psychological reaction. As, for example, in kleptolagnia, when a woman steals an article from a store in order that the emotional tension aroused by the act may procure a physical sexual reaction.

The psychological investigation of every adult found guilty of a crime is impracticable, and such investigation would be unprofitable in many cases. For although every crime is a mental problem involving a state of mind associated with certain conduct, it is not necessarily dependent upon obscure psychological reactions. Some persons favourably disposed towards the scientific investigation of crime are uneasy lest abstruse speculations concerning the causal factors of a crime supplant a clear conception of the criminal. They think with Putnam "that it is impossible to give a just impression of even the unconscious factors in the mental life unless one, at the same time, defines the influence of the conscious factors" [7].

In my view the psychological approach to crime is in danger from overstatements.

For example, it is sometimes suggested that every sex offender should undergo a psychological investigation in spite of the fact that a sex crime may be the normal reaction to a sexual invitation. At the same time, however, there can be no doubt that the psychological investigations carried out among adolescent delinquents in this country have been of material assistance in apportioning the most suitable treatment in individual cases.

Bernard Hart [8] reminds us that all methods of psychotherapy are dependent upon the employment of one or more of three basic principles, suggestion, persuasion, and analysis. He insists that every psychotherapist, although he may style himself a suggestionist, persuasionist or analyst, makes use of at least two and often of all three principles. Suggestion is omnipresent and will obviously come into action whenever there is doctor and patient. Many astute criminals have considerable powers of suggestion and persuasion also, and use them as weapons of defence. There is reason to believe that the conclusions resulting from psychotherapeutic conversations have erred sometimes because the law-breaker has been more successful than the doctor in the practice of these arts. Sometimes the offender does not accept the suggestion of the psychologist, but nevertheless uses it as an excuse for crime, particularly if the medical analyst seems to prefer an abstruse and speculative explanation for the offence when a simple one is more convincing to others.

Varying estimates are made in regard to the results of psychological treatment in criminal cases, but no substantial figures have been published on the matter as far as I know. W. Healy, who has directed much time and study to this question in America, is consistently guarded in his conclusions. H. E. Field [9] reports that at Sing Sing prison and the institutions under the Department of Correction at Massachusetts, psychology and psychiatry are regarded almost exclusively as a means of diagnosis and classification. The same inference is drawn from the observations of Sheldon and Eleanor Glueck in regard to the Massachusetts reformatory [10]. Indeed, this appears to be the usual rôle of the psychiatrist and psychologist in American prisons [11].

Richard C. Cabot has written [12]: "Psychologists, physicians, social workers and criminologists, who readily condemn the present methods of treating criminals, have themselves usually nothing to suggest but more accurate diagnosis. Character diagnosis is essential, and we lack it. But it is not treatment. . . . Psychiatry and psychology have given thus far few, if any, clues of proved usefulness in the treatment of crime."

Prinzhorn [13] estimates that definite cures by psychotherapy may be claimed in 20 to 30% of the cases. Recently Kessel [14] and Hyman have reported that the therapeutic results obtained by a competent and complete analysis of 33 patients showed bad results in 16, or 49%, including seven psychotics and the rest maladjustments of various kinds. In four cases the results were classified as good, and in 13 cases satisfactory specific results were recorded. There can be little doubt that law-breakers generally are difficult subjects for treatment, and if these figures are correct for civil cases, the successes in criminal cases will be still less. For even the first appearance of a law-breaker in a criminal court may be the result of a firmly established criminal habit and the sequel to a series of similar offences.

It is important to consider in this connexion the results, as far as can be ascertained, of the usual methods of dealing with offenders.

The results of the Probation of Offenders Act are instructive. At Liverpool, 246 persons were placed on probation during 1920, and 136, or 55·28%, reappeared at court during the next four years; 44·72% were successes. At Birmingham, 270 offenders were on probation in 1920; 95, or 35·1%, reappeared at court during the next four years; 64·9% were successes. At Leeds, of 145 persons whose period of probation expired in 1920, 24, or 16·5%, were brought again before the court during the next four years; 83·5% were successes [15].

The Cardiff authorities made an analysis of the results of the cases entrusted to their Probation Officers over succeeding periods of five years, and the analysis for the years 1919 (ten months), 1920 to 1923 shows that the percentage of persons on probation in Cardiff who, within a period of five years, again appeared before the court were respectively as follows: 1919, 44·82; 1920, 57·78; 1921, 47·82; 1922, 54·66; 1923, 41·78. The figures are based on all subsequent first reappearances, however trivial the charge, and the position therefore is probably better than the figures, 50% per cent. successes, would seem to indicate [16].

No statistics are available to show what proportion of those who come to prison for the first time do not return, but all persons who are experienced in prison administration agree that the proportion of first offenders who become reconvicted is very small.

The effect of military service on many habitual criminals during the Great War is of interest in this connexion. Men who appeared to be utterly incorrigible joined the Forces when released from prison and rendered useful service until demobilized. Under guardianship and employment, sharing advantages and disadvantages with their fellows, they became socially acceptable and useful members of the herd. Those of us who knew these men well cannot believe that psychotherapy would have proved so successful.

There is difficulty in following up the career of an ex-Borstal inmate over many years subsequent to his discharge if he is doing well. Letters may remain unanswered and visits may be resented if the subject considers that information concerning his past may come to the notice of his associates or employers if he co-operates in the inquiry. Of 609 persons discharged from Borstal Institutions for the first time in 1928, approximately 60% had not been charged in court again during a period of two years, and experience shows that the great majority of those who do return come into conflict with the criminal law again before they have been at liberty as long as this.

I have referred elsewhere to the fact that psychiatric examinations have been combined during recent years with psychological investigations in cases of adolescent offenders, and minor psychotherapy has been of direct value in the treatment of some cases [17]. The psychological approach has enabled some adolescent delinquents in Borstal Institutions to adjust themselves to social requirements under the guidance of the Borstal staffs. It is reasonable to expect that a certain number of mentally unstable lads will become stabilized in any case as they reach adult age in the sheltered life of an institution, perhaps more particularly if physiological imbalance has been faulty and is restored to normal requirements; and the extent to which the psychological investigation has aided the institutional training must remain a matter of conjecture in many cases. Experience shows that the investigation and the training may be of mutual assistance in the reconstruction of the individual.

The success of punitive measures in adolescence is largely due, like the psychotherapeutic, to suggestion and persuasion. The medical evidence submitted to the Persistent Offenders Committee pointed to the fact that the psychological investigation which was claimed to produce good results in delinquent cases was often no more elaborate than that which is open to social workers and probation officers, and is practised in Borstal Institutions.

It seems probable, however, that psychotherapy may succeed sometimes in cases where other methods have failed, because the psychotherapist is a more effective suggestionist or persuasionist and, also, may have better facilities to practise these arts,—sometimes because the skilled technique of a prolonged mental analysis is necessary. But there are dangers. An intensive psychological investigation is proposed sometimes in criminal cases when it is unsuitable, as in the case of a woman who murdered her child in consequence of a puerperal depressive psychosis,

although her chance of recovery at this time, soon after the murder, clearly depended upon the improvement of her physical health, and the rest that could be given to her mind. So, too, in the case of an artful offender who pretended to co-operate with the psychotherapist because he thought he was creating thereby a favourable impression and serving his own ends.

The effect of punitive detention on psychological treatment is held by some to be necessarily disadvantageous. This view seems to be founded on a misconception concerning the effect of imprisonment on law-breakers. A minority resent the measures of self-protection enforced by society; others may be unable to co-operate with the psychotherapist through apprehensiveness of what this may involve. But it is probably correct to state that the majority do not feel themselves to be unjustly detained, and are not hostile to the custodians who guard them or to the authorities who sentenced them.

However this may be, there will always remain a certain number of adult offenders whose crime is too serious and the danger to society too great to sanction their attendance at a psychological clinic as an alternative to imprisonment. In many cases the personality of the medical psychologist may be of more importance than the fact that the offender is serving a sentence of imprisonment, but the latter may be of curative value, and not a hindrance, if exploited by the suggestionist and persuasionist. So much so, that a flagellant who had undergone imprisonment as well as psychological treatment for this sexual deviation was unable to tell me which alternative had effected his readjustment, although he inclined to the view that the punitive measures had been most efficacious.

The Departmental Committee on Persistent Offenders reported that [18] :—

> "The primary aim of psychological treatment in criminal subjects is to prevent them from committing crime in future, and it is accepted generally that the fear of imprisonment is often more deterrent than the actual fact. We do not believe that a decision can be made at the present time between the relative advantages of psychological treatment prior to, or during the currency of, imprisonment. Different factors are involved in individual cases. Doubtless many offenders earnestly desire to be relieved of their anti-social tendencies. In others, mental abnormality may co-exist with malingering, an offender may consciously exaggerate his disability, and as we have already pointed out, may refuse to accept the truth if it threatens to abolish his means of self-gratification. One medical witness considered that the emotional reaction which followed upon a sentence of imprisonment placed the subject in a favourable mental condition for psychological treatment. But it seems probable that emotional reactions of an opposite character may have the reverse effect. Perhaps the prospect of future imprisonment may be the physician's most powerful ally in some cases, a sentence of imprisonment in others."

The difficult child has been described as the future potential delinquent, and the truth of this is apparent in many cases. But it is a partial truth only, and many criminals have led socially accepted lives during childhood and adolescence. There seems reason to hope that the psychological treatment of the abnormal child will sometimes prevent the development of criminal tendencies. The future may prove that in this lies the most practical contribution of psychology to the criminal problem.

Even if it is true, as I believe, that the scope of psychology in the treatment of crime will remain strictly limited in our present state of knowledge, we should persevere with our investigations and ascertain the most profitable type of case to submit for treatment, always remembering, however, that the medical psychologist owes a duty to the public as well as to the individual delinquent.

The experts who gave evidence before the Persistent Offenders Committee were of the opinion that the psychological investigation and treatment of crime might be most usefully applied in cases of homosexuality, exhibitionism, and obsession. I would suggest also in cases of some offences attributable to hysterical reactions.

Communal life is constantly increasing in complexity, and false deductions may affect the study of many social problems. It is contrary to all experience to consider that the so-called scientific treatment of crime is going to empty prisons, for although a psychological investigation may often determine the causal, environmental, and personal factors associated with social maladjustment, it may be unable to effect a readjustment, and it is probably correct to consider that the psychological approach to crime is more successful as a means of accurate diagnosis than as a method of treatment. It is suggested that it would be surprising if this was not so, for even if the psychological assumptions in regard to a particular case are acceptable, they do not explain why an individual reacts in a different manner to his fellows when subjected to similar influences. May not other biological approaches be more important, and perhaps more elusive than the psychological? From this point of view it would seem that often delinquent conduct may be due fundamentally to hereditary dispositions, which, when present separately in the parents, are not socially disabling, but may cause disharmony when combined in the personality of the offspring if they reinforce or antagonize each other when called into action by stress of circumstance.

There can be no doubt that certain judicial authorities would welcome the assistance of medical men when dealing with delinquents whose crimes are associated with mental abnormality, if assured that psychological treatment was a reliable alternative to imprisonment. Many other judicial authorities have doubts and misgivings, and there is reason to consider that this result may be due sometimes to the unrestrained optimism and assurance of certain investigators. Mental treatment is unable to keep pace with psychological theory and the general purpose of psychotherapy may be served best if only modest claims are made in regard to the treatment of crime by this agency. I am mindful that E. Mapother [19] has told us that he feels "that if psychopathology is to rise like other branches of biology from the anecdotal to the scientific level, and if psychotherapy is to become rational and to define its limitations, then uncontrolled clinical findings must clearly be supplemented by observations as to the effect of standard experiences under experimental conditions capable of repetition. At present there is a gross neglect of such experimental observations upon the varying responses to stimuli that may be noted or established in patients with different mental syndromes. Clinical tales do duty for demonstrable rules as to abolition of mental symptoms or even of experimentally conditioned responses resembling these in any way."

The difficulties which surround the scientific approach to the criminal problem are seldom fully appreciated. These remarks attempt only to indicate a few. The immediate problem is to ascertain how many law-breakers are genuine when they declare that they desire to be cured if a pathological condition is associated with the crime, and how many of these can be treated with success. To this end, a scientific investigation carried out at a penal institution, recording failures as well as successes, is much to be desired and is about to be undertaken.

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Section of Orthopædics

President—ST. J. D. BUXTON, F.R.C.S.

[*November* 7, 1933]

Four Cases of Fracture of the Os Calcis Treated by Bohler's Method.—C. HOPE CARLTON, F.R.C.S.

THIS method is an advance on any hitherto used.

Fracture of the os calcis is a severe injury, many of the patients never again returning to useful work. It is a common accident in the shipping industry through men falling down the hold and alighting on their feet, as a result of which there is a crush fracture of the os calcis. No worse treatment could be devised than simple immobilization of the foot in a plaster case. Before the introduction of Bohler's method I depended upon a tarsal arthrodesis to eliminate the pain at the surfaces of the fractured joints, of which the most important was the sub-astragaloid joint.

Before resection.

After resection.

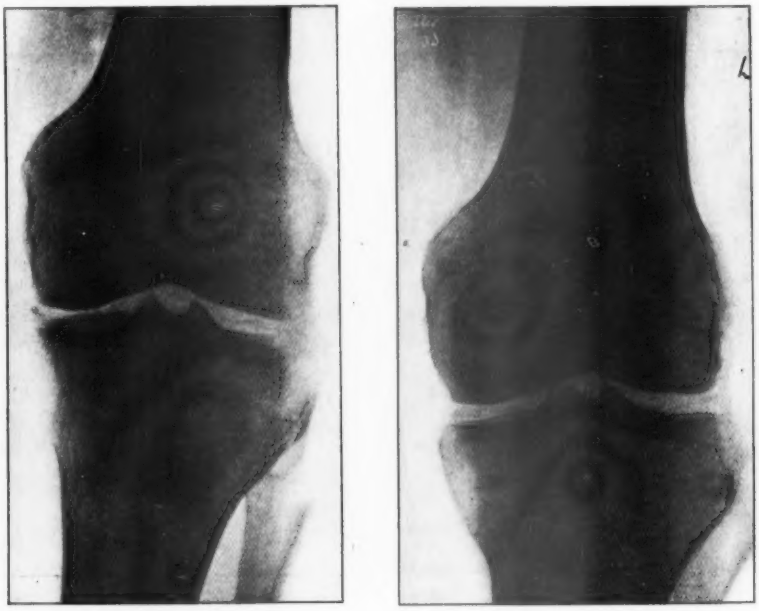
The method is described in Bohler's book ("Treatment of Fractures," 2nd ed., pp. 175-180). It consists in forcible traction of the bone in its long axis under anæsthesia whilst at the same time it is squeezed laterally by a special vice.

The four cases shown have all been seen marking time, a movement which calls for stepping down on the os calcis. All these men were injured as recently as the early part of this year. None of them has yet returned to his usual work, but it is to be remembered that such work is extremely arduous and consists in carrying baulks of timber and sides of beef in the docks. All four can walk at least two-thirds of a mile. Two are already employed in gainful labour of a less severe type than their usual occupations.

As the skiagrams show, there is not complete correlation between the clinical recovery and the recovery as shown by X-rays. In one case, that of which the skiagrams are shown here, the clinical result is as good as the final skiagram in

which it will be seen that the telescoping of the bone has been entirely pulled out. This case is shown by permission of Mr. Hugh E. Griffiths, under whose care it was. The other three cases were under my own care. After reduction as described, the foot is kept in extension with a pulley and weight for one month. Then a walking plaster with stirrup is used for from twelve to sixteen weeks, and after that a boot with an insole. Should there be any residual stiffness of the forepart of the foot, this is easily dealt with by manipulation under anæsthesia.

Calcification of the Semilunar Cartilages.—G. R. GIRDLESTONE, F.R.C.S. S. B., aged 46, came to see me at King Edward VII Hospital, Windsor, complaining of his right knee. He had twisted it in a cart-rut fifteen years before. This wrench soon cleared up, but following it he had had occasional sudden attacks of pain and swelling of the knee. They came on acutely, though not associated with



Calcification of the semilunar cartilages.

any injury, and lasted two or three weeks. He had recently recovered from one of these attacks.

On examination the knee appeared normal with the one exception that after sitting down for a few minutes, when he first got up and began to walk each weight-bearing movement of his knee was associated with a loud squeak. Incidentally his profession is that of a detective.

X-ray examination showed extensive calcification of both cartilages of the right knee, and a skiagram of the left knee showed a similar condition, though in a less degree. (See figures.)

Very few such cases have been reported, and so far as I have traced, none in this country.

A Family with Congenital Absence of the Heads of the Femora.—W. ELDON TUCKER, F.R.C.S.

(1) Mrs. P., aged 51. Patient has been told that her paternal grandfather was a cripple. Her first cousin on the paternal side was in Guy's Hospital continually with fragilitas ossium, up to the age of 14 years. Four of this cousin's children have suffered from fragilitas ossium.

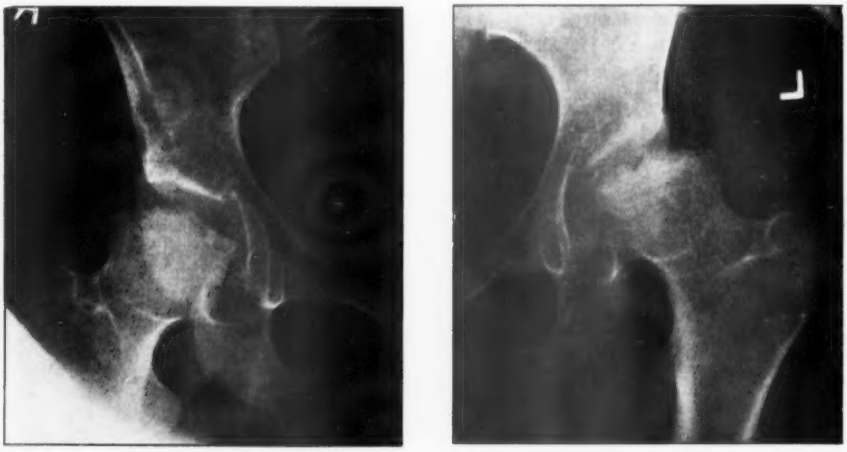
Mr. H. is Mrs. P.'s brother and Miss P. is Mrs. P.'s daughter. Mrs. P. has another brother with exactly the same deformity as herself and Mr. H. Mrs. P. has a son aged 24, who walks with a limp.

Mrs. P.

At the age of 12 years Mrs. P. began to have trouble with her hips and to walk with a limp. Only during the last five years has she had any pain. Now there is marked flexion-adduction deformity of both hips. The right hip is fixed in 20° flexion and 15° adduction. There is marked limitation of every movement in the left hip.

(2) Miss P., aged 15. This patient is supposed to have had rheumatic fever at 8 years of age. It has been noticed that she has been limping since that date, but that the limp has become much worse during the last two years.

The left hip is practically fixed in 15° flexion-adduction. There is a fair range of movement in the right hip, but each movement is definitely limited.



Miss P., aged 15.

Mr. H.

(3) Mr. H., aged 53, has experienced similar symptoms to those of his sister. His hips now are becoming so painful as to prevent work. There is marked flexion-adduction deformity, and there is only slight range of movement in both hips.

The following cases were also shown :—

Two Patients with Stiff Hips: Cases for Diagnosis.—J. M. GIBSON, M.B.

Two Patients with Infantile Paralysis, shown after Ramisectomy.—W. H. OGILVIE.

Mr. R. BROOMHEAD demonstrated a **Pneumatic Splint for a Fractured Clavicle.**

[*December* 5, 1933.]

Spondylolisthesis of the 4th Lumbar Vertebra with a Crush-fracture of the 2nd and Sacralization of the 5th.—S. ALAN S. MALKIN; F.R.C.S. (Edin.).

T. W., male, aged 50.

History.—On November 12, 1932, when first seen, he stated that six weeks previously he was crushed and buried by a fall of roof when working in a pit. He was taken into hospital and discharged at the end of a week. No skiagram was taken. Since then he had had constant pain and stiffness in the lower part of his back.

On examination he was found to have marked rigidity of the lumbar spine with pain on attempting flexion. There was no obvious deformity, but a skiagram showed spondylolisthesis of the 4th lumbar vertebra with sacralization of the 5th and a crush fracture of the 2nd. (See figure.)

Treatment.—Operation May 11, 1933. Long tibial grafts were placed on the rawed laminæ on each side of the spinous processes, extending from the 1st lumbar

vertebra to the sacrum. The patient was kept recumbent on a plaster bed for four months and was then allowed to get up, wearing a posterior spinal support. He was able to walk without pain and has continued to make good progress.

? Bilateral Intermetatarsal Bone.—S. ALAN S. MALKIN, F.R.C.S. (Edin.).

N. W., a girl, aged 11, was first seen on account of flat-foot. She was found to have an enlargement at the base of the first metatarsal bone. A skiagram showed an abnormality at the base of the metatarsal and an enlargement of the articular surface of the cuneiform. This case shows either an abnormality of the epiphysis

? Bilateral intermetatarsal bone.

of the hallux or an intermetatarsal bone; against the latter possibility is the fact that the bone is not placed dorsally.

According to Kohler, the intermetatarsal bone may articulate or unite with the first cuneiform or the first metatarsal and is usually dorsal in position. Its presence is very rare.

Dislocation of the Sacro-iliac Joint Reduced by Hoke's Traction.—R. BROOMHEAD, F.R.C.S.

The patient, a man aged 45, was working at the bottom of a 9 ft. trench on June 23, 1933, when the sides of the trench fell in and he was crushed and bruised. He was removed to the General Infirmary at Leeds where a skiagram (fig. 1) showed a

dislocation of the right sacro-iliac joint together with a fracture of the pubic ramus and some widening of the symphysis pubis.

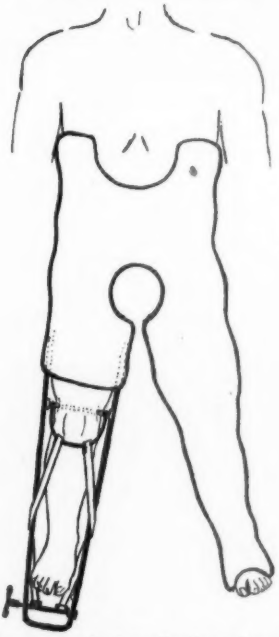
The man complained of pain round the lower part of the body, mainly in the back, but not of pain in the right leg or thigh, and apart from this pain he was well. He had a little difficulty in passing urine for a day or two though he had no retention.

The principle of "well-leg-traction" as devised by Hoke of Atlanta, is that as the sole of the foot is designed to bear weight, the sole of the sound limb should be made to act as the area for counter-pressure. The practical part of the method is easily understood when one considers that if the patient were standing on the left leg and one pulled on the right leg, the pull would be transmitted to the ground by the sole of the left foot. Accordingly, I suggested that we should apply skeletal

FIG. 1.—Skiagram showing upward displacement of right ilium (26.6.33).

traction to the right femur because the pull would be transmitted to the pelvis by the hip-joint ligaments and the ilium would be drawn into place.

The details of the method were as follows: A plaster spica was applied extending from, and including, the sound foot, to the nipple line and reaching nearly to the knee of the affected limb. The two bars of a Thomas knee-splint were incorporated in the plaster of the affected thigh, the bars being long enough to extend 6 in. beyond the heel of the affected limb. At the end of the splint instead of the usual V, a ratchet and pinion were fixed, and from the pinion to the U-shaped loop of the skeletal traction apparatus two bands of webbing were stretched (see diagram). We were thus able to use considerable force by means of fixed traction.



To show method of application of traction.

FIG. 2.—Skiagram with patient in plaster, showing upward displacement reduced but with wide space at joint (6.7.33).

On November 2, the patient could bend down and almost touch his toes; straight-knee knee-hip bending caused some pain in the right sacro-iliac joint, and he complained of pain and tenderness over the joint.

This case is interesting as showing what can be done by Hoke's method and also

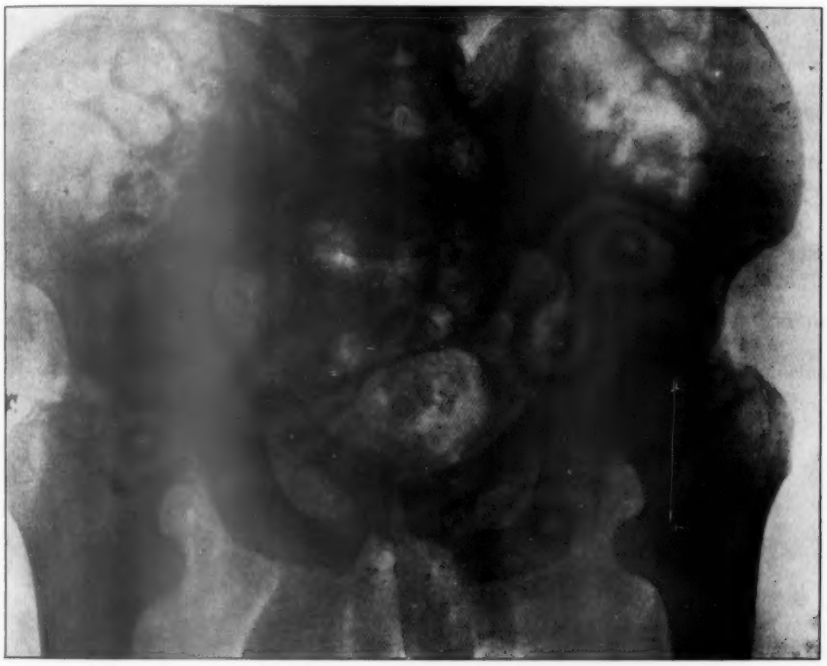


FIG. 3.—Skiagram showing displacement reduced and joint space normal (13.10 33).

because the patient has never had pain referred down the thigh from any of the nerves that supply the sacro-iliac joint, although we are told—and most of us believe—that in cases of sprain such pain is almost constant.

The patient can now bend down and touch his toes without difficulty or pain.

Traumatic Anterior Dislocation of the Hip.—E. J. SMITH, M.B., B.S. (for St. J. D. BUXTON, F.R.C.S., President).

S. D., male, aged 26, was giving an exhibition of weight lifting. The man's weight is 12 stone and he was attempting to lift 320 lb. above his head by the Terlazzo method (fig. 1), in which he leans forward to pick up the weight, swinging it up to shoulder level, and then lunges forward on his left foot to carry the body under the weight. At one stage of this procedure, the right hip is hyperextended as is shown. When in this position the right foot slipped, and the man collapsed.

Three hours later, when examined, he had little pain. The right lower limb was externally rotated but not abducted. The hip was slightly flexed; there was true shortening of about half an inch. A hard round swelling was felt externally to the mid-point of Poupart's ligament. This swelling moved with the femur, and bony

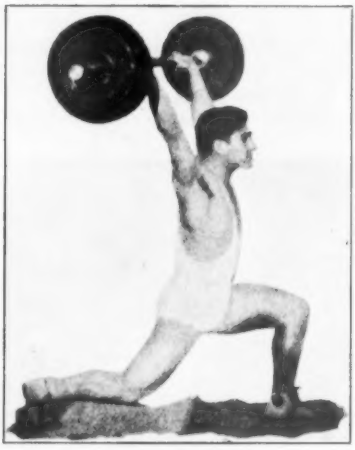


FIG. 1

FIG. 2.

crepitus could be felt. The great trochanter was displaced backwards; there were no signs of pressure on the common femoral vessels or anterior crural nerve.

X-ray examination showed anterior dislocation of the hip-joint, the head of the femur resting in front of and above the acetabulum, and a flake of bone torn off the upper lip of the acetabulum. (See fig. 2.)

Reduction was accomplished under general anæsthesia seven hours after the injury. The patient was placed on a mattress on the floor; the trunk was held down and the right lower limb flexed, abducted, and internally rotated while traction was applied manually. At the same time an assistant pressed the head of the femur downwards and backwards. Reduction was easy and full movements were obtainable at the hip-joint immediately after, with no tendency to recurrence of the dislocation.

X-ray examination following reduction showed that the detached flake of bone came from the upper lip of the acetabulum and was in satisfactory position.

After-treatment.—The legs were tied together for three days and then a plaster spica was applied.

Subsequent history.—Three months later the man was walking comfortably with full movement at this hip-joint, and was hoping to do weight lifting again.

Fracture of the Neck of the Femur, treated by a Modification of the Smith-Petersen-Pin Method.—B. H. BURNS, F.R.C.S.

I am presenting this case as an illustration of a simplified method of introducing a Smith-Petersen pin into the neck of the femur. The patient, aged 60, from a

FIG. 1.—Before operation showing typical displacement.

trivial fall four weeks ago, sustained a fracture of the neck of the femur with the typical displacement. Under spinal anæsthesia a Smith-Petersen nail was inserted by the method described below. Active movements were started after four days, and

FIG. 2.—Photograph taken during the operation showing the position of the Kirschner wire guides.

FIG. 3.—Showing the pin in position with the wires removed

she began to walk after a fortnight. She has now been walking with sticks for a fortnight. She has almost a full range of active movement and has no pain.

By using Kirschner wires as a guide, the introduction of the nail into the desired position is rendered much easier. The nails are specially made and have a hollow cylinder in the centre so that they can be threaded over a Kirschner's wire. A small incision is made down to the outer aspect of the great trochanter. Retractors are inserted giving adequate access to the bone, and the finger may be passed along the anterior surface of the neck. It is quite easy to palpate the fracture. The displacement is then reduced by an assistant internally rotating the limb and at the same time applying manual traction. Three Kirschner wires are now introduced in what appears to be the most suitable direction. They are then X-rayed in situ, and a film is developed as rapidly as possible. This takes about seven minutes. The wire which is best situated is selected and the other two are removed. Along the remaining wire the pin is then driven home. It is very easy to drive the pin too far if the measurement is directly assessed by comparing the pin with the skiagram, as this seems to magnify it considerably. If, however, a wire which is of equal length with the one partially embedded in the bone, is placed alongside it, the extra length will show how long the pin should be.

This is a minor operation and in my experience of twelve cases during the last four months, the shock is so little that it is applicable to every case of fractured neck of the femur. One of our patients was an old man, aged 84, who had an enlarged prostate. Unless guides are used it is difficult to be certain that the pin is driven into the correct position without performing an operation giving a wide exposure of the parts.

The following cases were also shown :—

Congenital Torticollis with Bony Abnormality of the Lateral Mass of the Atlas.—A. T. Fripp, F.R.C.S.

Lipogranulomatosis of Bone.—N. Capener, F.R.C.S.

Section of Dermatology

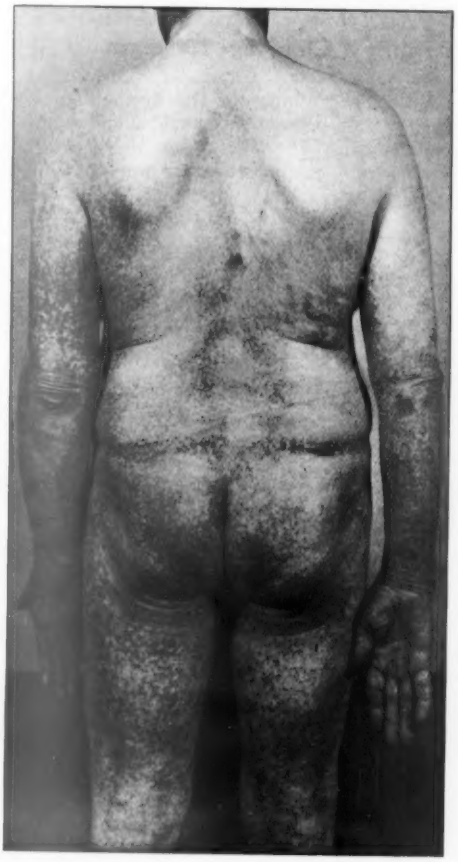
President—HENRY MACCORMAC, C.B.E., M.D.

[*December* 21, 1933]

Urticaria Pigmentosa with Telangiectasis occurring in an Elderly Adult.—J. E. M. WIGLEY, M.B., and GORDON HEGGS, M.D.

The patient is a man, aged 73. For fifty years he has been a warehouseman.

History.—Five years ago he first noticed this rash all over the trunk and arms. There was considerable itching at first. The rash consists of red, circular macular spots about the diameter of a lentil. It has never been raised above the surface



and after initial bright redness fades to a faint brown tinge. There is a marked reticular arrangement of the lesions. When first seen they appeared to be almost purpuric. The face, palms and soles are not involved.

Blood Wassermann reaction negative. Unna's friction skin test negative.

There are no symptoms other than backache due to an osteo-arthritis of the spine.

Histological report (Dr. I. Muende). The epidermis shows hyperkeratosis with a well-formed stratum granulosum. The capillæ are very widened. Blood-capillaries and lymph-spaces are markedly dilated. In the pars reticularis there are numerous cuboidal cells of an endothelial character containing refractile granules which take on polychromatic staining, with polychrome methylene blue stain. The condition is probably related on the one hand to "mast-cell nævus" and on the other to urticaria pigmentosa.

Dr. I. MUENDE said that when he first saw this case he thought the lesion was a healing lichen planus. He was surprised, on going into the histology, to find that there were numerous mast-cells; these, he considered, placed the case definitely in the group of urticaria pigmentosa. One feature which made it differ from typical urticaria pigmentosa was that the mast-cells were more cuboidal, more of the endothelial type; in fact, it reminded him of Dr. Goldsmith's recent case of mast-cell nævus, in which the cells containing the "mast-cell granules" were truly cuboidal.

Early Epithelioma in a Child aged 4 years.—J. E. M. WIGLEY, M.B., and I. MUENDE, M.B.

The lesion is a raised warty tumour, the size of a split pea, on the radial aspect of the right wrist. It has been present five weeks, and from its livid appearance suggests a verruca necrogenica. When first seen there was a hard painless gland palpable in the axilla, but this has since become impalpable. Otherwise the child appears to be perfectly healthy.

Histological report.—Marked acanthosis with a peculiar keratinization and attempts at concentric horny pearl formation, particularly in the lower parts of the rete pegs. At one part of the section the basal layer is very irregular and shows proliferating bands of epithelial cells growing into the corium. The upper parts of the corium are densely infiltrated with small round cells, a high percentage of which are of the plasma-cell type.

The histological appearances strongly suggest early malignant squamous-cell change.

Discussion.—Dr. I. MUENDE said that though the lesion was not a frank squamous-celled epithelioma, it was sufficiently evil-looking to suggest that it was pre-malignant; there was not the breaking away of the squamous cells, but there was present that heavy infiltration

of the corium which was seen in pre-malignant cases. The masses of plasma cells enabled one to rule out verrucose tuberculosis. In the latter there might not necessarily be giant cells, but the infiltration was usually of the lymphoid type.

The PRESIDENT said he was not convinced that the process was a malignant one. A pronounced plasma-cell infiltration was common in epithelial new growths of the skin but it might be interpreted in the present case as an inflammatory reaction to an infection, and the gradual disappearance of the gland in the axilla seemed to accord with this alternative diagnosis.

Pemphigus Vegetans.—H. MACCORMAC, C.B.E., M.D. (President).

E. A., an unmarried woman, aged 48, had facial paralysis when a girl, but otherwise has enjoyed good health. About ten months ago blisters appeared on the vulva, spreading to the left groin, and soon afterwards hypertrophic lesions appeared along the left inguinal fold. There have never been bullæ in the mouth or axillæ.

On admission a few bullæ were present in the groin, together with an elongated irregular solid mass with a rough surface: 6½ in. in length, about ½ in. across and 1 in. high. Parallel to this on the inside of the main tumour is a similar but much smaller lesion. There is a moist secretion with an extremely offensive odour. On culture a heavy growth of a diphtheroid bacillus together with *S. albus* have been obtained. It is to be noted that *B. pyocyaneus*, which is usually present in this condition, was not grown. Wassermann reaction negative. Blood-count: R.B.C. 5,220,000; Hb. 98%; W.B.C. 9,000. *Differential:* Polys. 51%; lymphos. 22%; monos. 3%; eosinos. 22%; basos. 2%. A section of the tumour shows marked acanthosis, pronounced dilatation of the blood-vessels and lymphatics in the corium, an infiltration of the dermis with polymorphs and large numbers of eosinophil cells, and several characteristic intra-epidermal micro-abscesses containing large numbers of eosinophils.

This case may be regarded as an example of the benign form of pemphigus vegetans. When the patient was first admitted to Middlesex Hospital the hypertrophic masses were treated with X-rays which, however, only slightly decreased their volume. In consultation with Mr. B. W. Windeyer it was then decided to implant

radium seeds in the lower half of the tumour mass and, if this proved successful, to treat the remaining portion later in a similar manner. In cases of pemphigus vegetans there is a special tendency to develop vegetating tumours upon any blister, for example, the blister produced artificially by carbon dioxide snow. In the endeavour to cure the patient it will therefore be necessary to treat the primary disease, and it is proposed with this object in view to give a course of germanin.

Discussion.—Dr. DOUGLAS HEATH said that during the last five years he had had two cases of this type, both in women aged about 55. One of these patients had recovered, and two years later had had a relapse. In her case the axillæ and groins were affected. The second woman had masses of the same type which extended from the axillæ down to the lower abdomen; the masses were about three-finger breadths, and their appearance exactly resembled those in the President's case, except that always in front of the advancing tumour there was a bleb-like edge at least half an inch wide from which streptococci could be cultivated. There was a high degree of eosinophilia. The angles of the mouth were affected, and round each angle vegetations developed. The Wassermann reaction was negative. No external treatment was successful by itself; he did not try either radium or X-rays, but immediately he gave 6 m of liquor arsenicalis, three times daily, she began to improve. When this was stopped the condition retrogressed, and when the treatment was resumed, improvement again set in. That confirmed the President's view that there was a basic condition. His (the speaker's) opinion was that such cases were more akin to dermatitis herpetiformis, in regard to response to treatment, than to true pemphigus. A case he had shown at a previous meeting of the Section was more comparable to Hallopeau's vegetating pyodermatitis, and the man had considerable masses all over his back, though there was no affection of either axillæ or groins. A diphtheroid organism was cultivated from the hands, body and mouth. After a long period of illness the patient recovered, and he (Dr. Heath) saw him many years later. He was unable to take arsenic. The best treatment to apply locally in these cases was calomel or xeroform in a drying powder. It controlled the fœtor to some extent and rendered the task of nursing less disagreeable.

Dr. S. E. DORE said he welcomed the placing of these chronic cases into a different category by the President and Dr. Douglas Heath, because this type of lesion differed from the rapidly fatal pemphigus vegetans. He had had a similar case,[1] which had previously been under Dr. MacCormac. The patient had enormous tumours in the groins and axillæ and on the buttocks, preceded by bullæ. These yielded an almost pure culture of *B. pyocyaneus* and improvement followed the administration of a vaccine made from that organism. The masses had eventually become so large that they had to be excised. The patient underwent two operations; after the first, the masses seemed to be healing up, but at the second she died under the anæsthetic.

Dr. W. N. GOLDSMITH said that he also had had a case which came into the same category as that now shown. The patient had been ill eighteen months before he saw her. There were thick masses of vegetating tissue in the nostrils and outside the nose, which was about five times the normal size. There were similar lesions on the eyelids, round the mouth, on the finger, and occasionally on the vulva. They began with, and were generally surrounded by, small blisters. The net result of many bacteriological cultures was that the blisters were sterile. The patient had a moderate eosinophilia and this was noted also in the exudate from the blisters. She responded astonishingly to arsenic, which was given by injection. It was remarkable to see how the lesions melted away when one reached a certain degree of arsenical medication, only to relapse when that treatment was stopped. Eventually, however, arsenic had to be abandoned owing to nausea and diarrhœa. Germanin was then given; after about the third injection there was an eruption at the site of injection—first, of superficial blisters like the other lesions, which were sterile, and then beneath these a large fixation abscess developed. The pus was sterile but contained a large preponderance of eosinophils. From that time the patient began to improve, and had continued to do so. He had heard, from time to time, that she was still well.

Dr. A. M. H. GRAY said he had wondered whether the present case was of the type which was included by some under the term "dermatitis vegetans," as distinct from pemphigus vegetans. What was the relation of this to the Hallopeau cases? He (the speaker) had

[1] *Brit. Journ. Derm.*, 1929, xli, 408.

inherited from the late Dr. Radcliffe Crocker one of the genuine cases of the Hallopeau type, the case which was published by Dr. Pernet in the *Journal of Cutaneous Diseases* (1912, xxx, 517). In that case the lesions were mainly confined to the back; there were huge plaques of vegetating dermatitis over the centre of the back, and the lesions were certainly primarily pustular. The whole vegetating area was studded with pustules, and the growing edge had a pustular margin. This patient was in hospital several times, and a large staphylococcus was cultured. Dr. Crocker had treated her with a vaccine obtained from this growth. This had always cleared up the eruption; and when the patient came to consult him (the speaker) the condition again cleared up under the same treatment. It would also be interesting to know whether the condition dermatitis vegetans was merely a variety of pemphigus vegetans, whether it was the same as Hallopeau's condition, or whether all those rightly belonged to the same group.

Poikilodermia of the Face (Civatte's type) in a Girl aged 14½ years.—F. PARKES WEBER, M.D., and O. B. BODE, M.D.

The patient is a well-grown girl, aged 14½, who for the last two years has had a blotchy red (telangiectatic) and white area on each cheek (symmetrical), to which since the commencement of November 1933 some superficial brownish pigmentation has been added over the red portions. Menstruation commenced at the age of 10 years, but ceased after an appendix operation at the age of 11½ years. After that operation she became increasingly fat till she weighed 13 st. 12 lb. in May 1933. Her menstrual periods then commenced again (May 1933), and have remained regular till now. In May a course of treatment was commenced with thyroid substance, prolan, and an ovarian follicular preparation, and she lost 2 st. in weight by November 1933. The blood-serum gives negative Wassermann and Meinicke reactions.

The red, white and brown colouring of Civatte's poikilodermia was very characteristic in November, and the patient (as in some other cases) has certainly had a considerable endocrine disturbance. But, after the continued use of a weak salicylic acid ointment to the poikilodermatous areas on the face, the brownish portions (? epidermic pigmentation) are now (at the time of the meeting) much less marked.

Dr. R. T. BRAIN said he wondered whether it was considered sufficient for the diagnosis of poikilodermia to take a condition in a child so young which did not show all the features described by Civatte—the telangiectasia and the pigmented reticular pattern—which characterized the cases arising at about the time of the menopause; it was the latter association which gave rise to the idea that the basis of the skin condition was some endocrine disturbance. In 1930 he (the speaker) had shown to the Section two cases of Civatte's type;[1] he thought those were different from Dr. Parkes Weber's case, particularly as the lesions began peripherally at the sides of the face and tended to creep inwards; smaller lesions were present on the forearms and legs. In those cases, too, a little superficial atrophy could be recognized.

Psoriasis Arthropathica in a Woman whose Twin Sister was Similarly Affected.—F. PARKES WEBER, M.D.

The patient, W. H., an unmarried English woman, aged 31 years, has typical psoriasis on the hairy scalp, the trunk (especially about the umbilicus, vulva and anus) and toes. The psoriasis has troubled her for the last five years, and during that time she has occasionally had articular stiffness, sometimes accompanied by joint swelling, in one or more parts (ankles, feet, spine, shoulders, elbows, knees). With the articular attacks the psoriasis has generally been less marked, and once in connexion with such an attack it cleared up completely for a time.

In other respects the patient, who is well developed, appears to have enjoyed good health. Her blood-count and urine show nothing abnormal. Her finger nails

[1] *Proceedings*, 1930, xxiii, 1481 (Sect. Derm., 43).

and the nails of both her big toes have sometimes been involved. Recently both her big toe nails were shed, and the nail of the little finger of the left hand is uneven.

Family history.—The patient's twin sister, who resembled her closely, suffered from the same skin and joint trouble, but more severely and from an earlier age (from about 22 years of age). She died at 25 years (? pneumonia). Patient's mother, who has never suffered in the same way, is living, aged about 60 years. Patient's father, who was likewise never affected in the same way, died of pneumonia at 43 years. There was no consanguinity between her parents. Of her nine other brothers and sisters, eight are living and one died of pneumonia as a young man, but none of them has had any cutaneous or articular disease, excepting a sister (Ivy H., aged about 25 years). This last sister has had psoriasis from the age of 2 years, but very little articular trouble.

Discussion.—Dr. H. SEMON said that he had had a similar case three years ago in a man, aged 35, who had been in hospital under the physicians for months. There was arthritis with the same succulent type of psoriasis. The patient was bed-ridden, and attended the out-patient department in a chair. He (the speaker) had put him on large doses of salicin, i.e. 15 gr. three or four times a day, and the improvement that followed was extraordinary; in three weeks the joints were less painful, and the psoriasis began to disappear within six weeks from the beginning of treatment; the condition was unrecognizable as psoriasis. He wondered whether this type of psoriasis was associated with intestinal toxæmia.

Dr. A. M. H. GRAY said that it was important to get the patients in such cases to bed; they improved more quickly in hospital than if treated outside. This type of case appeared to improve on intravenous injection of salicylate of soda, though that treatment seemed to have little effect on the ordinary case of psoriasis. He had seen more cases of acute psoriasis—particularly those with associated joint symptoms—improved by this method of treatment, combined with rest in bed, than by any other; and it seemed that the improvement was not due to intestinal disinfection.

Dr. NORMAN BURGESS said that Dr. Wills and he had shown a similar case at the Annual Meeting of the British Association of Dermatology in 1931. The patient had been bed-ridden for some time on account of arthritis and also had generalized psoriasis. Intravenous injections of novarsenobillon were given, and he made a complete recovery. He had now been back at his work for eighteen months, and there had been no recurrence either of the arthritis or of the psoriasis.

He (Dr. Burgess) had found that arthropathic psoriasis particularly affected the nails, and he asked whether this was the general experience.

[Dr. ROBERT KLABER'S case of **Necrobiosis Lipoidica Diabeticorum (Urbach-Oppenheim)** will be published with a coloured illustration in the next issue of the *Proceedings* of the Section.]

Section of Radiology

President—R. S. PATERSON, M.D.

[*November* 17, 1933]

DISCUSSION ON THE IMPORTANCE OF POSTURE IN RADIODIAGNOSIS

Dr. E. W. Twining: *Posture in radiography of the skull.*—This paper deals with posture in relation to the diagnosis of intracranial tumours, and falls naturally into two divisions: (1) Direct evidence of tumour. (2) Evidence obtained by ventriculogram or encephalogram.

DIRECT EVIDENCE

If we exclude the pituitary adenomas, the localization of which is obvious, the intracranial tumours of surgical importance which are most likely to show direct radiographic evidence or change in the skull wall are the meningiomas. These important tumours, superficial in origin, non-invasive, and amenable to surgical removal, can be divided into two groups: (1) those which occur in the vault, and (2) those of the base.

Meningiomas of the vault.—The parasagittal meningiomas spring from the meninges near the superior longitudinal sinus. The tumours of the convexities arise somewhat further from the mid-line. These two together give direct X-ray signs in about two-thirds of the cases. A third type, meningioma of the falx, hardly ever produces erosion of the skull.

These tumours cause erosion or hyperostosis, or both together, of the vault over their point of attachment: the area of bone affected is usually small in proportion to the size of the tumour: hyperæmia of the overlying bone is a characteristic finding, and is evidenced by porosis, and by enlargement of the diploic veins. These altered diploic veins, which have obviously enlarged to cope with the intense hyperæmia over the actual tumour, often present peculiar characteristics in the radiogram. They pursue a somewhat direct course downwards to the nearest emissary area. They are rather uniformly widened, with parallel sides, and in this they differ from normal diploic veins, which show narrowings and enlargements along their course. They remind one, with their sharp-cut edges and rather direct course, of the bed of a flooded torrent on a mountain side, whereas normal veins suggest rather the sluggish streams and backwaters of the plains. To this type of enlarged vein I give the name "purposive" (fig. 1). Such a vein is sometimes the only localizing sign of a meningioma: and the point I wish to make is that it is often easier to decide on which side it lies by stereoscopy than by an antero-posterior in which it is hidden; I mention the point because some very experienced radiologists question altogether

FIG. 1 (Case 1).—Enlarged diploic veins in a case of meningioma.

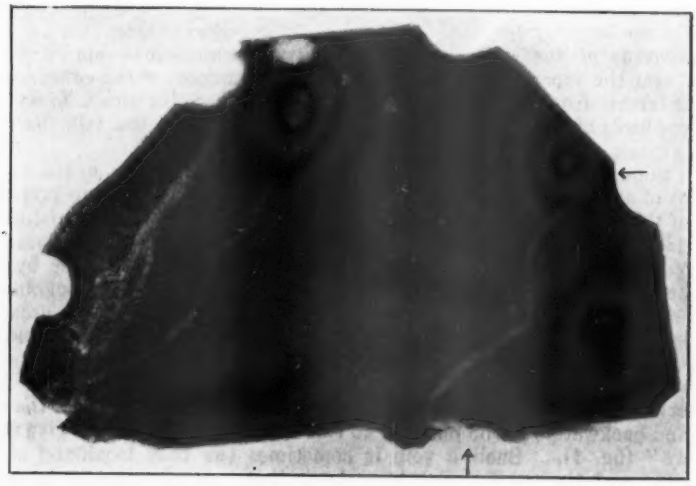


FIG. 2 (Case 2).—Meningioma. Radiogram of skull-flap. Arrowed: altered diploic vein which lay over tumour.

the value of stereoscopic skull films. The skull-flap of another case, of which fig. 2 is a radiogram, shows well the character of the "purposive" diploic vein, compared with diploic veins of a more normal type. Although this vein could not be seen in the postero-anterior view, its position in stereoscopic radiograms before operation accurately localized the tumour, a huge parasagittal meningioma in the posterior parietal region.

Bone erosion and hyperostosis.—The point to remember is that the affected area is usually small in proportion to the size of the tumour and suspicious regions have to be carefully examined stereoscopically and tangentially. Particularly in the case of hyperostosis of the outer table, a tangential view of the diseased area is essential to demonstrate the typical fluffy spicules on the surface of the vault over the point of the attachment of a meningioma. A localized thickening of the inner table ("enostosis") which sometimes occurs over a meningioma, also requires a tangential view, to bring out its essential character. The enostosis shown in fig. 3 by means of a tangential view, accurately localized a meningioma which was much larger than might have been anticipated from so small a bone lesion. This tumour, successfully removed by Mr. Jefferson, was 3½ in. in diameter.

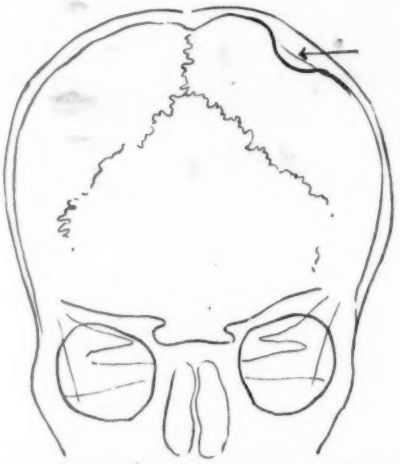


FIG. 3 (Case 3).—Hyperostosis of inner table. Meningioma.

Meningiomas of the base (fig. 4).—These also arise in the neighbourhood of the venous sinuses. They have been grouped according to their common sites of origin. I want to draw special attention for our present purpose to: (1) The olfactory groove meningioma; (2) the sphenoid ridge tumour and sylvian cleft meningioma; (3) the tumour arising from the back of the petrous, occupying the cerebello-pontine angle, and simulating clinically and radiologically an acoustic neuroma.

(1) *The olfactory groove meningioma.*—This tumour, lying far forward in the skull, is accessible to the surgeon, but relatively inaccessible to X-ray, because its point of origin is on the irregular and thin floor of the anterior fossa, and because it may produce very little in the way of signs. Half of them produce X-ray changes. But if the signs are slight, positions will have to be devised to show them.

Ordinary lateral views may show some thickening of the floor of the anterior fossa—not very striking or obvious, but enough to arouse suspicion. A film is then taken which is not a true lateral, in order to show one orbital roof above the other.

This may show a small tuft of bone formation on one side, near the ethmoidal groove. It is of advantage in such cases to make the stereoscopic shift up and down the couch instead of across it, so as to separate the floors of the anterior fossæ of the skull from one another in this manner.

A third method, sometimes useful, is a postero-anterior with the head flexed, in order to show the sphenoidal roof and the anterior clinoids, a position similar to Granger's position for the sphenoidal sinus. It shows the anterior and posterior clinoids, and, by demonstrating erosion of the anterior clinoid on one side, or bony

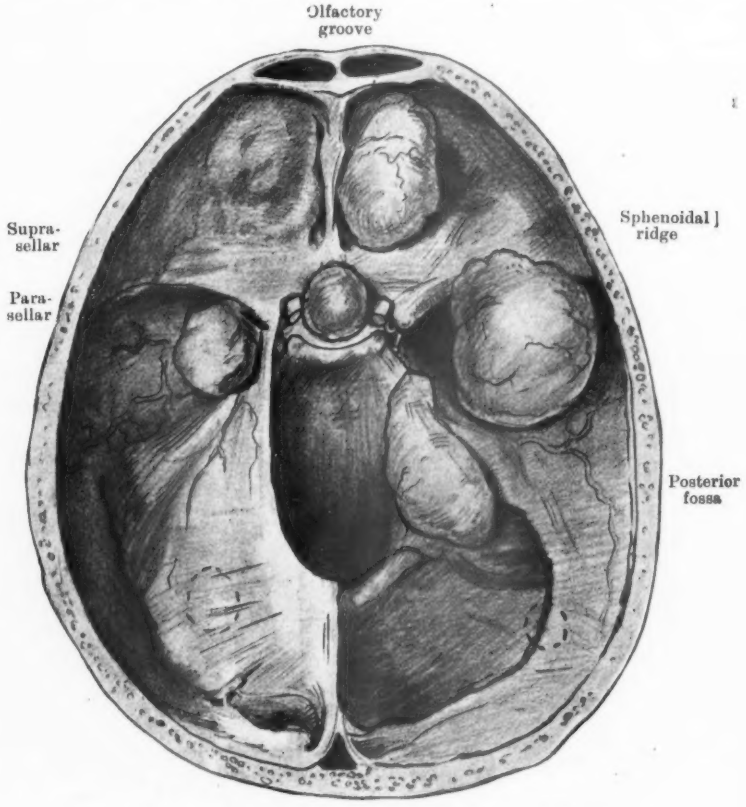


FIG. 4.—Diagram showing sites of some meningiomas of the base.

changes near the mid-line in the anterior fossa of the skull, may localize an olfactory groove meningioma or other tumour of this region.

A view which is occasionally useful for olfactory groove meningioma is the Rhese position for the optic foramen, because it shows so clearly the ethmoidal roof as well as the optic foramen and the anterior clinoid. Incidentally, meningiomata as well as gliomata of the optic nerve may dilate the optic foramen; but they seem to be very rare, for though we have had many suspected cases referred from the Eye Hospital, I have never succeeded in finding dilatation of the optic canal by such a tumour.

What I have said of olfactory groove meningiomas holds good for orbito-ethmoidal osteomas, though these are usually easier to detect.

(2) *Meningiomas of the sphenoidal ridge*, arising from the lesser wing, may cause extensive hyperostosis of the middle fossa and of the lesser wing itself. About two-thirds of these tumours produce direct X-ray signs.

The lesser wing can best be studied either: (1) by lateral stereoscopics, shifting the tube across the couch in order to separate the lesser wings; or (2) by projecting the lesser wings into the orbits by a modification of the "chin-nose" projection.

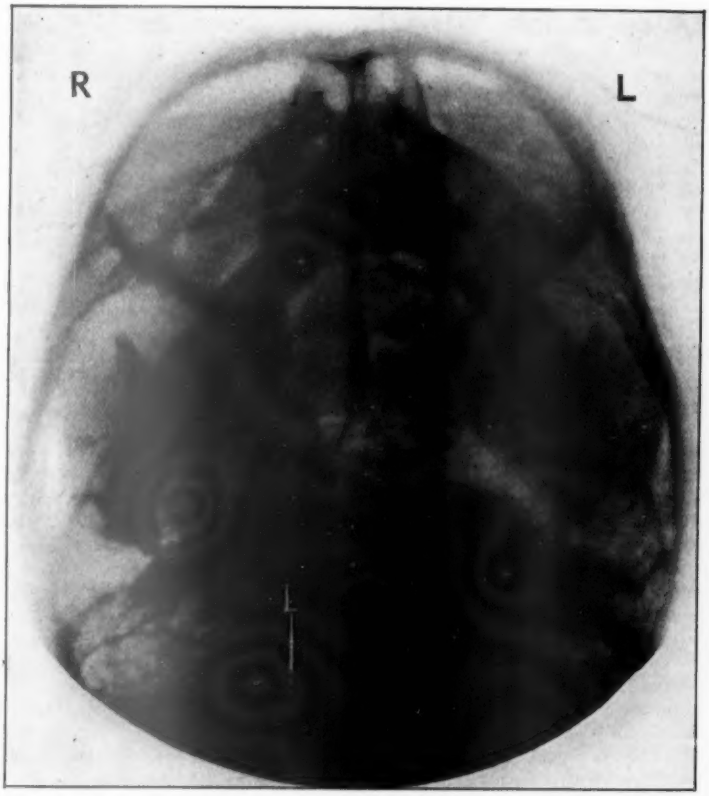


FIG. 5.—Vertical projection. Destruction in middle fossa involving foramen ovale. Endothelioma.

Either hyperostosis or rarefaction of the lesser wing may occur. Rarefaction of the lesser wing has been extensively studied by Olivecrona and Lysholm, and is not very helpful. It may be misleading, for instances are recorded in which the lesser wing opposite to the tumour has been more rarefied than that on the tumour side, and this has led to false localization.

Destructive changes in the middle fossa itself are best studied in the vertical projection. Fig. 5 shows destruction of part of the base, due to an endothelioma of the pharynx invading the middle fossa from below. It had destroyed the left foramen

ovale and a wide area behind it and to its outer side. I have not seen a case of such destruction of the foramen ovale by meningioma of the middle fossa, though this may occur. Extensive hyperostosis of the middle fossa and temporal bone is the more common sign of the meningiomas (usually "en plaque") of this region.

(3) *The meningiomas of the posterior fossa* are difficult to localize. They produce direct signs in only about one-third of the cases. Caution should be exercised in attributing to tumour the convoluted diploic veins sometimes seen in the lateral view below and behind the petrous bone. These are normal. The most useful localizing sign is erosion of the petrous bone. This sign is also produced, of course, by the much commoner acoustic neuroma, and by some rare tumours of vascular origin, and we may consider them together. The question is, which is the best method of showing erosions on the posterior surface of the petrous bone.

(*a*) *Stenver's position* shows the bone well, but superimposes anterior and posterior surfaces, and is therefore unsuitable for our purpose.

(*b*) *Projection of the petrous bones* (postero-anterior or antero-posterior) *into the orbits* allows of a comparison of the densities of the two petrous bones. It shows the lip of the internal porus, the internal auditory canal and the semicircular canals fairly well, but it does not show the back of the petrous bone.

(*c*) *Vertical projection.*—If large, an erosion on the posterior surface of the petrous bone may sometimes be shown in the vertical projection, but generally speaking this projection is disappointing.

(*d*) *The internal auditory meatus* can be brought into line with the external, using a projection similar to Schuller's (lateral, head elevated 15°, and rotated towards the tube 10°) for the mastoids. The method was devised by Henschen of Jena, in 1910, for detecting enlargement of the internal meatus by acoustic neuroma. Cushing used it in his clinic, but found difficulty in exactly duplicating the correct position, and for this reason it was given up. The difficulty in getting the exact position is easily understood when we consider how frequently the postero-anterior view discloses asymmetry of the petrous bones, and of the direction of the internal auditory meatus.

I have found that the correct position for superimposition can most easily be obtained by screening the patient through a small diaphragm and tilting his head until the internal porus shows visually as a bright translucent dot through the external auditory meatus of the same side. This is surprisingly easy to do, and enables a very close comparison of the internal pori to be made. [Examples were shown of enlargement of one internal porus demonstrated by this method.] But I have practically given up this method as a routine, for two reasons: (1) Some acoustic neuromata do not dilate the meatus, but merely erode superficially its opening on the back of the petrous bone with the surrounding bone. In this case the superimposition method is valueless; (2) because a much better method is available. This is a method of fronto-suboccipital projection (fig. 6). The patient lies on his back with the chin well flexed so that the orbito-meatal line is vertical. The film is placed with its centre in the suboccipital region. The central ray makes an angle of about 30° with the orbito-meatal line and grazes the posterior surface of the petrous bone. (Fig. 7 taken in this position, shows well a posterior erosion of the right] petrous bone due to meningioma in the cerebello-pontine angle.) [Other examples of erosion of the petrous bone demonstrated by this method were shown.]

This projection I have for many years found useful for occipital fractures and for localizing small tumour calcifications in the posterior fossa which were hidden by the petrous bone in the normal postero-anterior view, but since I learned from Dr. Sachs of Baltimore that it had been described by E. B. Towne in connexion with acoustic neuroma I have always used it for this purpose.[1] It is called "Towne's position" by American writers, and is, without doubt, a most useful method.

1 *Archiv. Oto.-Laryngol.*, 1926, iv, 515.

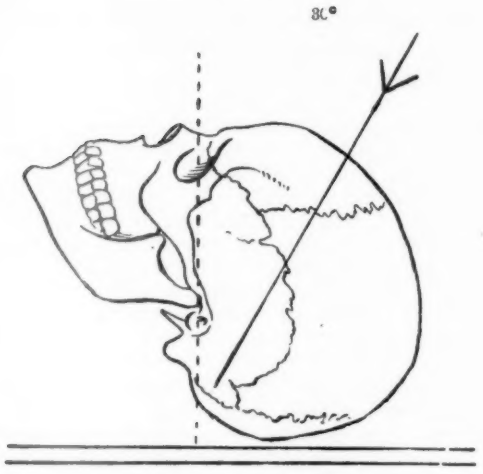


FIG. 6.—"Towne's" projection.

FIG. 7.—Erosion of right internal porus. Meningioma. Towne's projection.

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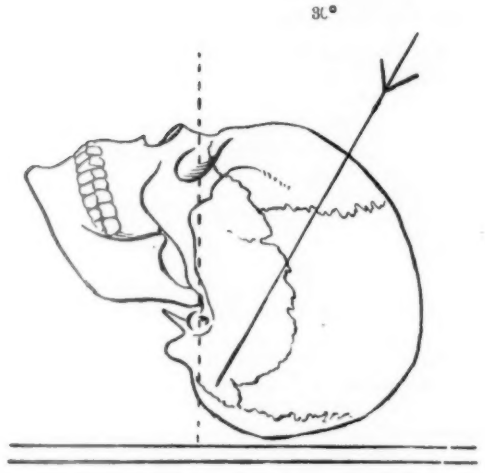

FIG. 6.—"Towne's" projection.

FIG. 7.—Erosion of right internal porus. Meningioma. Towne's projection.

VENTRICULOGRAPHY AND ENCEPHALOGRAPHY

In ventriculography and encephalography, interpretation of the radiograms depends upon a knowledge of the posture in which they are taken, and this part of my subject provides many instances of useful variations of posture.

We may consider: (1) Investigation of internal hydrocephalus often, of course, due to tumour; (2) investigation of pressure deformities of the ventricles due to cerebral tumours and atrophy of brain tissue.

Hydrocephalus in children and adults.—The cerebrospinal fluid is secreted from the choroid plexus in the lateral ventricles and flows via the foramen of Monro, third ventricle, the aqueduct of Sylvius, the foramen of Magendie and the foramina of Luschka into the cisterna magna, then forwards through a chain of basal subarachnoid cisternæ to the region of the Sylvian fissure to reach the surface of the brain where it is continually absorbed. There are certain points of narrowing where a block is easily caused—foramen of Monro, third ventricle, aqueduct and the foramina of Magendie and Luschka. We have examples of blocks at each of these points, but in children the two commonest are cerebellar tumour and pachymeningitis in the posterior fossa. In adults acoustic neuroma and suprasellar cyst and mid-brain tumour must be added. The resulting internal hydrocephalus causes expansion of the brain, which, in children, produces its greatest effect at the vault, causing the characteristic gaping sutures, convolutional impressions and globular skull. In adults these signs are far less marked and may be altogether absent, and in them a very marked hydrocephalus often exists with no bony changes in the vault whatever: but there is one point on which the dilated ventricles can exert a maximum influence on the skull wall, i.e. at the base of the brain where the third ventricle lies close to the pituitary fossa. The third ventricle dilates and forms a thin-walled sac which presses first on the posterior clinoid processes, eroding them, and then on the pituitary fossa which it distends and destroys.

Sellar destructions due to pituitary or suprapituitary tumour cannot always be distinguished from those due to hydrocephalus, but much information may be obtained by means of ventriculography.

Ventriculography in a case of obstructive hydrocephalus involves the use of a good many special postures. First of all, what is the problem involved? Put simply it is this. At what position is the obstruction? If it is in the region of the third ventricle it will require a frontal or temporal surgical exposure; if in the mid-brain, it is likely to be irremovable; if in the posterior fossa it will require sub-temporal surgical approach. So that the aim of ventriculography in such a case is to demonstrate the site of obstruction if anterior, or by exclusion to limit its probable site to the posterior fossa.

Patency of the foramina of Monro.—This can be tested very graphically on the fluorescent screen with the patient sitting up. If he bends his head forward and turns it to one side we can cause a transference of air and cerebrospinal fluid at will from one side to another, until the fluid levels are at the same height. Usually it is enough to note whether the third ventricle fills, and whether air passes across from the ventricle which has been injected to one which has not. There are certain surgical tests which render this radiological investigation unnecessary in most cases.

If the third ventricle fills we want to know if it is dilated. If it does not fill we want to know why.

A dilated third ventricle is sometimes well shown in the antero-posterior view below and between the lateral ventricles. It is still better shown in the vertica projection. The central ray runs directly through the anterior end of the third ventricle, and the radiogram will show the anterior ends of the lateral ventricles with the third between them. In the lateral view, if the ventricles have been completely emptied, the whole of the third ventricle is sometimes seen. But in a

case of obstructive hydrocephalus one very rarely gets such good emptying. Some fluid remains, and in the lateral position excludes air from the third ventricle. In order to make sure of filling it, and demonstrating its exact shape and outlines, I devised some years ago the following technique. The patient lies on his back with head extended as for a vertical projection, but instead of the usual view, a lateral is taken (fig. 8). The air rises into the anterior end of the third ventricle and distends it, showing us two projections with a notch between. The notch is due to the optic chiasma. The projection behind it is the infundibulum of the pituitary body. Fig. 9, made by this method, shows how the dilated third ventricle reaches down to the sella, which it has destroyed. Obviously this method has possibilities of detecting tumours above and in front of the sella, which deform the third ventricle, and I have already met with examples of such deformity due in one case to craniopharyngioma, in another to suprasellar meningioma, and in a third to meningioma of the base.

If in addition to tilting the head back, the couch is tilted as well, we can by this method fill the third ventricle as far back as the opening of the aqueduct, and show the pineal recess at the posterior end of the ventricle. But we cannot show the

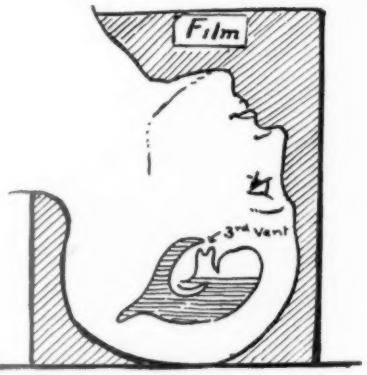

FIG. 8.—Ventriculography. Special view for third ventricle.

aqueduct and fourth ventricle. The fourth ventricle can occasionally be shown by encephalography, air being introduced by lumbar puncture with the patient sitting up.

In two cases of suspected pineal tumour I have examined in the position shown in fig. 10. It is a prone position with exaggerated flexion of the head. It shows the cleft between the lateral ventricles in which deformity by pineal tumour might be expected, but in neither of these cases was pineal tumour present, and no deformity was found.

Apart from the investigation of cases of obstructive hydrocephalus, which may be due not only to tumour, but to adhesive meningitis, we have to consider deformities of the ventricles resulting from lesions of the cerebral hemispheres. In this case information can usually be gained by a routine set of films, one antero-posterior, one postero-anterior, and a pair of laterals, but it is often necessary to devise, on the spot, modifications of these, or special positions to show the tumour more clearly. With parietal and temporo-sphenoidal tumours the deformity and lateral displacement of the ventricles is usually easy to recognize in antero-posterior and postero-anterior films. Tumours of the frontal lobe push one or both anterior horns backwards and

FIG. 9.—Third ventricle shown as in fig. 8. Third ventricle dilated and eroding sella. Hydrocephalus.

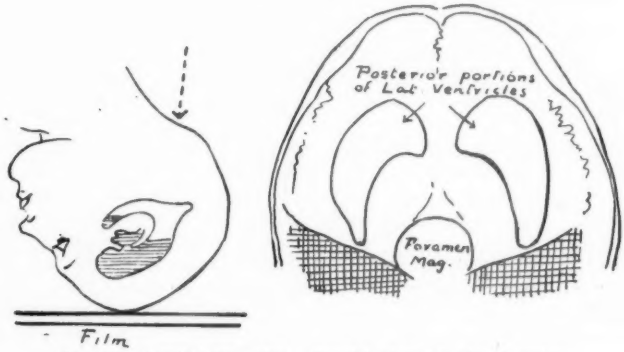

FIG. 10.—Ventriculography. Special view for pineal region.

produce an indentation of the anterior contour (fig. 11*a*). It is not always easy to say on which side the tumour lies because the deformity is not easily appreciable in the antero-posterior film, and in the laterals it may be very difficult to determine the side of the lesion: we find stereoscopy of little help. A method of identifying the right and left horns in such a case consists in the following procedure. The patient lies supine in order to keep both anterior horns filled with air, and then rotates the head alternately slightly to the left and then slightly to the right. In each case a lateral view is taken in this position (fig. 11*b*). Rotation towards the tube brings the ventricle nearest to the film uppermost; rotation away from the tube brings the ventricle further from the film uppermost. By comparing the two films it is easy

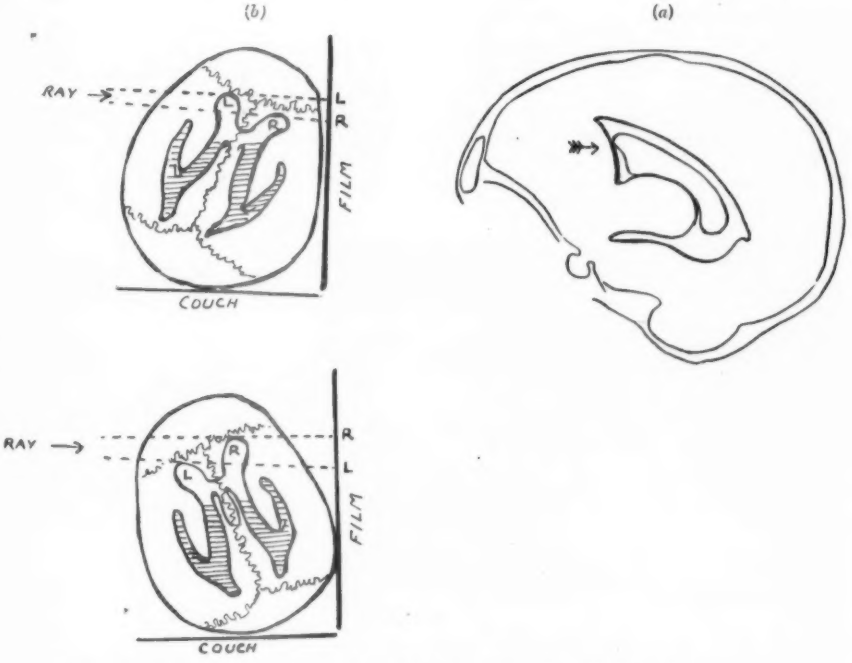

FIG. 11.—(*a*) Ventriculogram. Right lateral, lying. Filling defect in frontal horns. (*b*) Ventriculogram. Right lateral, lying. Filling defect in frontal horns. Patient lies supine, film to side of skull. Forehead is rotated towards and away from film.

to identify the right and left horns respectively and to show which is the more deformed of the two. In one case this was the only guide the surgeon received as to the side of the lesion, and enabled him to cut out his flap on the correct side.

Routine laterals.—It should be noted that the shape of a partly air-filled ventricle in lateral ventriculograms with the patient lying, differs greatly with position. If that ventricle is lowermost, air collects in its middle portion—because the ventricles are arched towards the mid-line here. If it is uppermost, air collects at its extremities —the frontal, occipital and temporal horns; so that we may get a pseudo-filling-defect of the extremities of the lowermost ventricle, and a central pseudo-filling-defect

in the uppermost (fig. 12, *a* and *b*). Unless this is appreciated a false diagnosis of tumour may easily be made, and it is essential to study each radiogram with the most careful reference to the position in which the head was placed when it was taken. The filling-defect arrowed in fig. 12*b*, at first sight resembles the tumour filling-defect in fig. 11*a*, but is solely due to posture.

Antero-posterior.—In this position the frontal horns, being uppermost, are filled. They lie near the mid-line, separated by the septum lucidum, shown as a dark median

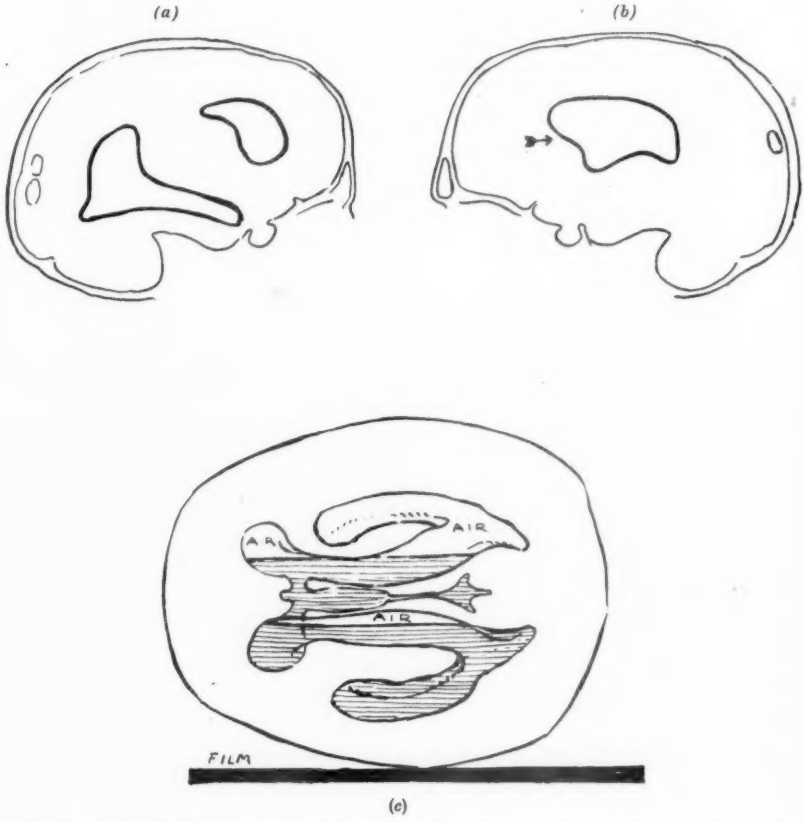

FIG. 12.—Effect of posture on partly filled lateral ventricle. (*a*) When uppermost, the ends are filled. (*b*) When lowermost, the central portion. (*c*) Explanatory diagram.

shadow, and with the third ventricle form the "ventricular butterfly." It is essential that the head should be flexed, so as to throw the frontal sinus below, and clear of the ventricles. In this position deformity of the ventricle by a parietal tumour, and displacement of the whole system to the opposite side is well shown.

Postero-anterior films, face downwards, show the posterior portions of the ventricles filled with air, and because they diverge from the mid-line we get a different picture, resembling a flying vulture.

A *lateral* view, with the patient face down, shows the posterior horns well, and demonstrates or excludes deformity due to tumour.

This position gave an interesting finding in a case of atrophy of the occipital lobe following an injury. In the radiogram one posterior horn was normal—the other reached to the occipital end of the skull where the ventricle and the skull-wall are practically in contact, as a direct result of total local atrophy of the occipital cortex.

The examples given do not illustrate fully the subject of posture in relation to ventriculography. Nearly every case presents its own special problem, and requires a postural technique specially adapted to that problem.

Dr. J. V. Sparks: *Posture in radiography of chest cases.*—The posture of the patient is primarily of importance in the routine examination, because it is from these views that we must decide if any subsequent examination is to be of help.

The posture which I employ for the routine anterior view is one in which the patient lies prone on a cassette tilted at an angle of about 10° towards the apices, with the forehead resting on a sandbag; the elbows are flexed and the shoulders are pressed well down and forwards (fig. 1). I have adopted this position as the standard because it gives the closest apposition between the patient and the film, and because

FIG. 1.

it throws the scapulæ and breast shadows clear of the lung fields, thus giving the best definition and detail in the lung. I emphasize the importance of this posture for the routine view because the diagnosis of tuberculosis depends on the recognition of tubercles in the X-ray film, so that every possible means must be utilized to obtain such detail in the films that these tubercles may be apparent.

Naturally, the first variation from this view is the routine erect anterior view which I take with the patient sitting, and not standing, as many patients are able to sit up who might not be able to stand. From an examination of these two views,

a decision can usually be arrived at as to whether any subsequent examination is necessary. Such may include a posterior view, a right or left oblique view, or a right or left lateral view. In addition to these views, or as a substitute for any one of them, there are certain cases which may require special examination in unusual positions, by reason either of the condition of the patient or of the appearances seen.

When special views are demanded by reason of the condition of the patient, it is often necessary to be content with a posterior view, and where the presence of fluid is suspected and the patient is too ill to be placed in the upright position, fluid can often be demonstrated by turning him on to his side and taking an anterior or posterior radiogram with the rays passing horizontally parallel to the floor (fig. 2). The fluid level, of course, only appears in cases which would normally show a level in the erect position, such a level depending on the association of the fluid with air.

FIG. 2.

There are special views which may be of value in certain cases, and which require a careful screen examination in order to obtain the correct position. These include views which are taken with varying degrees of rotation of the patient to demonstrate any particular lesion noted on the screen. Such views are employed for the localization of pleural adhesions prior to thoracoscopy, the localization of abscess cavities prior to operation, and for the localization of encysted fluid prior to paracentesis. It is only natural that the presence of fluid in association with air should give the greatest opportunity of determining the value of posture. It is usual for such fluid to obscure the base, and it can easily be displaced from this position by inclining the patient at such an angle that the fluid will flow into a different part of the pleural cavity. Such a view is of use to show the base in cases where there is a hydropneumothorax. The patient may be tilted to any angle, or even completely inverted.

I do not propose to make any reference to special apical views, as the routine radiogram should give as perfect detail here as elsewhere in the lung fields, provided that the film is tilted towards the apices to obtain the closest apposition. I should, however, like to refer briefly to Dr. Rowden's technique for showing abnormalities at the base, since basal abnormalities are far more liable to be overlooked, because the lungs extend downwards for a considerable distance in front of, and behind, the visible contour of the dome. To obtain this view of the base the patient is placed on the film in the supine position, as for the routine posterior examination, but the tube is centred over the frontal bone. This position brings into view the lung posterior to the dome, as far down as the eleventh rib, and similarly one can, by turning the patient into the prone position and centering over the occiput, bring into view the lung anterior to the dome.

In my opinion we shall learn a good deal more about the progress of disease in the lungs when these special views are utilized more often.

Dr. G. B. Bush : *Posture in radiodiagnosis of the abdomen.*—I propose to confine my remarks to various points which arise in the X-ray examination of the alimentary tract and the gall-bladder, dealing quite briefly with some points, and at slightly greater length with a few that I think will be of major interest.

There are two factors which, it seems to me, render the question of posture of vital importance in the examination of the abdominal viscera. One is that these organs can be considerably altered in their shape and position by a change of posture (an aspect of human anatomy which has been so brilliantly dealt with and stressed by Barclay). Moreover, in the evolution of man from the lower animals, the change of posture from the horizontal to the vertical has resulted in modifications of the abdominal viscera and their supports which are of importance in the consideration of the subject of visceroptosis. The other factor is that most of these organs are hollow cavities or tubes, and we rely, for the radiological demonstration of their anatomy, physiology and pathology, on a radio-opaque fluid to fill them, and fluids themselves are necessarily influenced by any alteration in posture.

Now we can use the force of gravity to assist us in many of our diagnostic problems when making use of opaque fluids. This force is constantly opposed in the body by the force of muscular tone, exerted both in the organs themselves, and also in the muscles of the abdominal wall. The force of gravity can be kept under the control of the examiner, while on the muscular tone of the patient's organs he can have little or no influence. For the exercise of this constant control over the force of gravity during an examination, making it a most helpful servant instead of a frequent source of inconvenience or difficulty, I have found a simple motor-driven tilting couch, fitted with a Bucky diaphragm, and over- and under-couch X-ray tubes, a most valuable piece of equipment. Moreover, a good deal of otherwise necessary palpation can be dispensed with, and a thorough examination of the patient can be made in the minimum of time, and with the minimum of fatigue to both the operator and the patient.

Let us now consider, as briefly as possible, a few applications of these principles to the study of the alimentary tract and gall-bladder.

Œsophagus.—In the examination for strictures, it is usual to watch the swallowing of an opaque medium with the patient erect. If films are made thus, one can show the *site* of the stricture, but its length and extent are much better shown with the patient lying down and on his side.

Cardio-œsophageal junction.—This region is often very difficult to examine

satisfactorily. It is easy to miss pathological and other abnormalities here unless variations of posture are employed as a routine. The type of case which comes to mind is that in which there is a slight hernia of the cardiac end of the stomach through the diaphragm, or a congenitally short œsophagus with partial "thoracic stomach." In the former case, the cardia may not lie above the diaphragm when the patient is erect, or only very slightly so, and the gas bubble may be too small to be noticed behind the cardiac shadow, but in the supine position the state of affairs will be more clearly manifest.

Early carcinoma of the fundus of the stomach it is also possible to miss, unless the patient is screened in every position. The supine position with very little barium generally shows it best.

Stomach.—A motor-driven tilting-couch is particularly valuable in the examination of this organ. A small quantity of barium can be made to swill over the entire mucous membrane from fundus to pylorus, and watched under the screen with the greatest of ease and rapidity in the majority of cases, while it is possible to make film records showing every part filled by posturing the patient in the inclined position, at about 30° to 40° to the horizontal. This is particularly the case when examining an atonic flabby stomach. In the erect posture the fundus is usually empty and the lower pole filled, and often the meal falls away from the pylorus and duodenum unless some form of compression is used, while in the horizontally supine position, the meal flows up to the fundus, which lies much nearer the patient's back, the pyloric end being close to the anterior abdominal wall and remaining empty or only poorly filled.

An inclined supine posture renders the long axis of the stomach horizontal, the angle varying with the type of patient, and I have found the position most valuable for both screening and film records of posterior wall and lesser curve lesions, using only a small quantity of barium.

Duodenum.—There are many ingenious devices for demonstrating the duodenal bulb, and alteration of posture plays an important part. Here, again, a tilting-couch is most useful. The condition known as duodenal ileus, often associated with visceroptosis, is of interest in dealing with the subject of posture. One comes across a fair number of these cases, generally in young women, and it is difficult to assess the importance of the phenomenon as a cause of symptoms. Duval, Roux and Béclère lay considerable stress on it in their symposium on "The duodenum."

I am inclined to think it only of importance, firstly, if I can find definite evidence of organic obstruction in the third part of the duodenum or just beyond, uninfluenced by posture, and secondly in those cases in which the duodenal delay is definitely influenced by a change in posture, and there is a fairly large gastric and duodenal residue after six hours. The first class is surgical and comparatively rare, while those cases falling within the second class I am accustomed to designate by the term "postural duodenal ileus," and to grade them into two classes as "moderate" or "severe," according as the obstruction is relieved by a change of posture from erect to supine or prone. In the severest cases only the prone position will relieve the temporary obstruction; in the moderate cases the meal passes fairly easily in the supine and the prone position, while in all of them the condition is most marked in the erect posture. An anatomical subdivision is also made according to the site of the temporary obstruction. In all the cases falling into the second class, it is probable that the factor of mechanical obstruction is of much less importance than that of deficient or ineffectual gastric and duodenal peristalsis, and in those cases in which there is no gastric delay I think the causes are more likely to be "functional" than mechanical, and the condition is not inconsistent with health.

A fairly comprehensive discussion of the whole subject has been published by

Zeitlin,[1] who says that he has studied the phenomena of duodenal motility in some 2,000 cases. He arrives at somewhat similar conclusions.

Jejunum and ileum.—In cases of obstruction of the small intestine, it is unnecessary to stress the importance of examination in the upright position in order to demonstrate fluid levels. As these patients are often very ill, a motor-driven tilting couch is again most helpful, for the patient can be placed on a stretcher on the couch, and raised nearly to the vertical to demonstrate these fluid levels, with no expenditure of energy on his part. (The same applies to cases of fluid collections in the chest when the patient is too ill to exert himself.) The vertical position is also necessary when there is free gas in the peritoneal cavity, and, again, in many cases of diverticula.

With regard to the colon, the question of posture is of importance, (*a*) in determining the degree of mobility in cases of visceroptosis and (*b*) in deciding the question of fixation of any of its sections.

I have only time for a few words about the pelvic cæcum. The cæcum tends to lie deep in the pelvis (1) when the ascending colon is longer than usual; (2) when the cæcum is excessively mobile or there are congenital maldevelopments of the mesentery; (3) when it is fixed by adhesions from inflammatory processes in the female pelvis. I have found the following manœuvre useful in persuading the pelvic cæcum to ascend into the right iliac fossa where it can be palpated. The patient is tilted into a slight Tredelenburg position, and turned slightly on the right side to relax the right abdominal muscles, and pressure is exerted obliquely downwards from the left side. This pressure squeezes coils of small intestine down into the pelvis from the left side if the patient's bladder is not too full, and these coils push the cæcum up, the force being redirected from the pelvic floor.

Before I conclude with a short consideration of the examination of the gall-bladder, I must refer briefly to *visceroptosis*. This, of course, is a subject in itself, and I will do no more than suggest that there are still abroad many misconceptions on the subject. Its evolutionary significance has already been mentioned, but I strongly suspect that we may be wrong if, on demonstrating nothing more than a degree of visceroptosis in a patient radiologically, we jump to the facile conclusion that this in itself is the cause of the symptoms on account of which he or she has been referred to us. It is more probable, I think, that the visceroptosis is only one link in the chain of cause and effect, and that there is an underlying condition—nervous, or metabolic, or both—which could be more truly described as the major cause of the symptoms complained of.

Gall-bladder.—Soon after adopting a motor-driven tilting couch to effect change of posture in the routine examination of the alimentary tract, I noticed in one or two cases that gall-stones were more clearly demonstrated on Bucky films taken in the vertical position than on those made horizontally. I then investigated a case of typical gall-bladder colic, using the oral method of cholecystography, and found that not only was the gall-bladder itself more clearly shown vertically, but that cholesterin calculi, which in the horizontal position spread out in a thin layer over the anterior surface of the dye-filled gall-bladder and were almost invisible, sank to the bottom in the vertical position and formed a distinct layer with a horizontal upper level, and could be easily distinguished. A sceptical surgeon informed me that this was impossible, as gall-stones floated, so a test was made with an excised gall-bladder, bile, and gall-stones which were radiographed, both with and without added dye, in the vertical and horizontal positions. The test clearly proved that gall-stones tend to congregate together at the bottom of the gall-bladder, and are more likely to be demonstrated in the erect than in the horizontal position. I eventually discovered a case of multiple opaque small gall-stones, a slide of which,

[1] *Radiology*, 1933, xxi, 337.

taken in the two positions, demonstrates very clearly the points under discussion. Minute calculi and collections of gall-bladder "sand" can sometimes be shown in this way in the course of a cholecystographic examination, and I now make horizontal and vertical radiograms as a routine.

Much of interest and importance has yet to be stated about the influence of posture in the X-ray examination of the genito-urinary tract and of the spine and pelvis, particularly in spinal curvatures, and lumbo-sacral conditions, such as "sacro-iliac slip," but enough has been now said about the importance of posture to indicate the immense interest of this aspect of radiodiagnosis and to stimulate further research into the mechanism of the living human body both in health and disease.

Section of Anæsthetics

President—H. P. FAIRLIE, M.D.

[*February* 2, 1934]

MODERN VIEWS ON RESPIRATION

Normal Regulation of Respiration

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Respiratory centre [1].—The respiratory centre probably extends throughout the pons and the upper part of the medulla oblongata. Trans-section of the midbrain in animals has no effect on breathing, but if the upper part of the pons is damaged the respiratory rhythm becomes markedly altered. Inspiration is now very deep and may be prolonged for two minutes or more; the inspiratory muscles are so violently contracted that it is difficult to expel the air manually from the chest. The inspiratory muscles then relax, at first gradually, but finally suddenly and considerably, expiration thus taking place. The cycle then repeats itself. When the section is through the upper border of the medulla the type of breathing just described (which is called by Lumsden "apneustic" breathing) is replaced by a regular series of gasps. Trans-section at the level of the apex of the calamus scriptorius permanently abolishes all rhythmic respiration.

To explain these phenomena, Lumsden has postulated the presence of centres in the lower brain stem, each of which is released if the centre above it is destroyed. In the upper medulla, below the striæ acousticæ, is a *gasping centre* which, when working alone, causes gasping respirations. Just above the level of the striæ acousticæ is the so-called apneustic centre, which is responsible for the prolonged inspirations. In the region of the upper pons is a centre which normally checks the activity of the apneustic centre and so produces normal respiration. The nerve-cells having this effect he calls the pneumotaxic centre. There is no need, however, to adopt this artificial subdivision of the central grey matter. Normally the "centre" acts as a co-ordinated unit; if parts of it are cut off, the portion left can no longer control breathing normally, and so abnormal types of respiration are produced.

Cases have been reported in infants in whom, following birth injuries, apneustic breathing has been observed; post mortem, hæmorrhages in the pons could be demonstrated [2].

When an animal is dying from asphyxia, overdose of anæsthetic, or interference with the medullary blood supply, it may be noted that as the breathing fails, it sometimes passes through the stages of apneuses and gasps; if the animal is revived, breathing recovers in the opposite order.

Rhythmic activity of the respiratory centre [3].—The respiratory centre is connected with, and controls the activity of, the lower motor neurones which supply the muscles of respiration. There is little doubt that the respiratory centre possesses an inherent rhythmicity, analogous to that of the sino-auricular node of the heart. Under experimental conditions it still goes on discharging in a rhythmic manner after it has been isolated as far as possible from afferent impulses from the lungs, the circulatory system and the higher centres and after the respiratory muscles have been paralysed by means of curari (Adrian).

Direct recording of the electrical changes in the respiratory muscles shows that in cats and rabbits under chloralose anæsthesia the expiratory muscles may be regularly in action during the expiratory phase of the cycle; in the decerebrate preparation, and in animals under dial and urethane, they are occasionally in action, but never so in deep chloroform anæsthesia. The expiratory muscles, however, always contract when the depth of respiration is increased.

The depth of breathing is mechanically regulated in a number of ways. It is well known that if skeletal muscle is stimulated at low rates it goes into partial tetanus and develops a low tension ; if stimulated at sufficiently high rates, it becomes fully tetanized and develops its maximal tension ; further, the tension of a muscle will vary with the number of its fibres that are in action simultaneously. Appropriate electrical studies [4] show that during quiet inspiration, the diaphragm, for example, is being stimulated through its motor nerve at low rates (e.g. 25 per second) ; the tremulous state that might be anticipated from such incompletely maintained contractions is obviated by the various fibres of the muscle not all being in the same phase of activity simultaneously. In deep breathing a larger number of fibres come into action. They are stimulated more synchronously and rapidly (up to 60 or 100 per second) to give a full tetanus and the duration of the contraction may also be prolonged. If the respiratory centre is damaged or poisoned it is found that the nervous impulses reaching the respiratory muscles become more infrequent and of shorter duration ; inspiration thus becomes feebler and briefer, and finally may cease completely.

Regulation of the breathing [5].—A brief consideration of the purposes served by breathing forms the most satisfactory introduction to an account of its regulation.

(1) The self-evident reason for breathing is to obtain supplies of oxygen and get rid of carbon dioxide formed in the body. Our oxygen requirements and the amount of carbon dioxide liberated are proportional to the degree of activity of the body. The first law of breathing is therefore (all other things being equal), that the pulmonary ventilation *is proportional to the metabolic rate.* It can be shown that for every calorie of heat liberated in the body, about 200 c.c. of oxygen must be used. Every 100 c.c. of inspired air provides about 5 c.c. of oxygen to the blood : it follows that a minute oxygen consumption, of say 200 c.c., demands a pulmonary ventilation of 4 litres. In a man of average size and weight, the heat output under basal conditions (i.e. at complete rest from twelve to eighteen hours after a meal) is about 1·2 calories per minute, necessitating a ventilation of 5 litres. The above considerations show how the resting ventilation can be calculated in any individual. Any condition which alters the rate of metabolism, e.g., thyroid disease, exposure to cold, changes in body temperature, the taking of food, decompensated heart disease, etc., will all correspondingly modify the resting ventilation. In muscular exercise the oxygen consumption (and metabolic rate) may increase up to 10- or 15-fold or even more, necessitating a ventilation of 50 up to 100 litres per minute.

The link between metabolism and pulmonary ventilation is probably provided by alterations in the carbon dioxide tension in the blood, to which the breathing is exquisitively sensitive.

(2) The breathing responds in an extremely delicate manner to the slightest change in the hydrogen-ion concentration of the blood in such a way as will keep the reaction at its normal level. The second great principle to remember is that the pulmonary ventilation will vary with the *reaction of the blood.* This matter will be considered in greater detail later.

(3) The breathing is sometimes adjusted to *maintain the normal temperature* of the body. When the body is exposed to warm and very moist external surroundings, regulation of body temperature becomes very difficult as heat loss by evaporation and radiation from the skin is greatly interfered with. The pulmonary ventilation under these conditions is greatly increased in order to eliminate more water vapour in the expired air and thus get rid of heat from the body. The alveolar and arterial carbon dioxide tension falls, but the alkalæmia resulting is compensated for in the usual way by the excretion of an alkaline urine.

(4) Changes in the *level of the arterial blood-pressure* modify breathing reflexly ; a rise of pressure depresses breathing and a fall stimulates it [6]. This factor probably only comes into operation in the intact resting individual when the blood-pressure is altered rapidly and to a considerable extent.

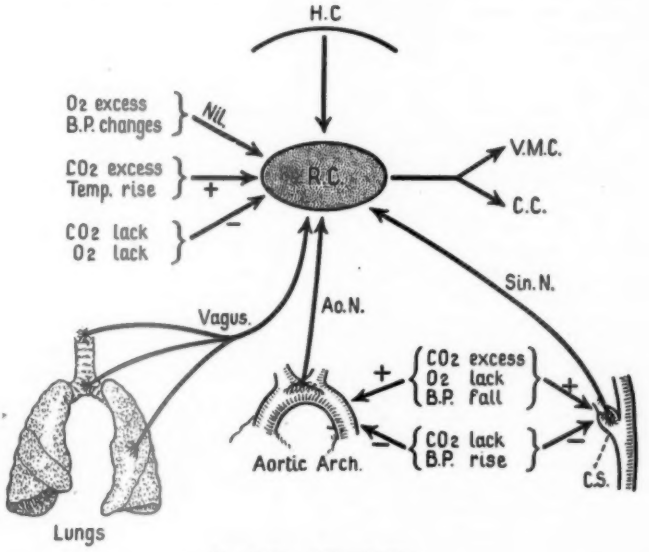
(5) Under certain conditions breathing is adapted to *maintain the appropriate oxygen tension in the blood.* We shall see that breathing only responds to oxygen lack when it is severe. At sea-level when air of normal composition is inhaled, oxygen lack can play little or no part in controlling breathing. At high altitudes, or in oxygen-poor atmospheres, it becomes the dominating factor and stimulates breathing, although there is no change in metabolic rate, and although the effect of the hyperpnœa is to produce an alkalæmia. The goal of the respiration under these circumstances is, of course, to improve the oxygenation of the blood.

(6) The *rhythm* of the breathing is mainly controlled by the pulmonary afferents in the vagi.

(7) The *sino-aortic nerves* which arise in the carotid sinus (a dilatation at the bifurcation of the common carotid artery) and the aortic arch play an important part in regulating the ventilation owing to the fact that their sensory nerve-endings in the blood-vessels are affected by changes in the pressure and chemical composition of the blood [7].

(8) Lastly it should be remembered that the respiratory mechanism is definitely depressed in *sleep*, and the breathing becomes inadequate for the needs of the body.

We must now proceed to a more detailed discussion of some of the factors just referred to. The chart below summarizes most of the information to be considered.



Regulation of Respiration.

R.C.—Respiratory centre. H.C.—Higher centres. C.C.—Cardiac centre. V.M.C.—Vasomotor centre. C.S.—Carotid sinus region (including carotid body.) Sin.N.—Sinus nerve. Ao.N.—Aortic nerve. + Stimulates breathing; − depresses breathing.

Carbon dioxide and breathing.—If air containing excess carbon dioxide is breathed, the ventilation is markedly increased, first by increasing the depth, and later by also increasing the rate of breathing. There are considerable individual variations in the response. When the carbon dioxide percentage in the inspired air exceeds that found in alveolar air (5·6), adequate compensation becomes impossible and carbon dioxide is retained in the blood. Headache, diminished sensibility, weakness and mental

confusion develop, and later loss of consciousness may occur. Finally, respiration is depressed and the circulation may fail chiefly because of poisoning of the heart.

If the carbon dioxide tension of the blood is artificially lowered by over-ventilation, breathing is depressed and may stop. Following the apnœa, periodic breathing may develop. The effects of over-ventilation also vary considerably and in a proportion of subjects it appears to be impossible to arrest the breathing no matter how prolonged the period of hyperpnœa may be. If pure oxygen instead of air is inhaled during the period of forced breathing the duration of the apnœa is greatly prolonged and in one recorded case lasted for eight minutes. If the over-ventilation is carried out with air containing more than 5% carbon dioxide, apnœa cannot be induced, as the blood carbon dioxide is not lowered below the threshold level.

The other effects of over-ventilation in man are worth mentioning. There is leucocytosis, hyperglycæmia, diuresis (possibly due to increased blood-flow through the kidney resulting from splanchnic vaso-dilatation), increased excretion of phosphate, and the usual urinary changes to compensate for an alkalæmia, i.e. a more alkaline urine and lessened excretion of ammonium salts. Acetone bodies appear in the urine; this may mean that aceto-acetic and oxybutyric acid resulting from fat breakdown are passed into the blood to help preserve its reaction. The blood-pressure is little altered, in spite of depression of the vasomotor centre, possibly because of the intense constriction of the skin vessels which is usually noted; the skin is cold and white.

Mode of action of carbon dioxide [8].—Recent research has shown this to be of a complex character. Carbon dioxide acts mainly directly on the respiratory centre, but it also acts partly reflexly by stimulating sensory nerve-endings in the carotid sinus region and the aortic arch, thus sending excitatory impulses to the centre. After section of the sinus nerves carbon dioxide excess may produce a smaller increase in the ventilation than in the intact animal.

Hydrogen-ion concentration and breathing.—Changes in hydrogen-ion concentration of the blood due to causes other than increased carbon dioxide formation in the tissues or to excess carbon dioxide in the inspired air readily affect respiration, in order (as previously indicated) to preserve the appropriate blood reaction. Thus if the hydrogen-ion concentration is increased (acidæmia), the breathing is stimulated, and extensive amounts of carbon dioxide are eliminated from the lungs and blood; the amount of carbonic acid in the blood is thus lowered and the hydrogen-ion concentration is consequently reduced to its proper level. Conversely when the blood reaction becomes excessively alkaline (alkalæmia), the breathing is depressed, carbon dioxide is retained in the blood and the consequent increase in H_2CO_3 helps to restore the normal reaction.

Common causes of acidæmia are (i) starvation, diabetes, or unbalanced carbohydrate to fat ratios in the diet leading to accumulation of acetone bodies: (ii) the ingestion of ammonium chloride which liberates hydrochloric acid in the body; (iii) methyl-alcohol poisoning (owing to the formation of formic acid; (iv) certain types of nephritis from retention of non-volatile acids. Alkalæmia may occur clinically, (i) from ingestion of excessive amounts of sodium bicarbonate; (ii) in pyloric or high intestinal obstruction and some cases of diarrhœa and vomiting. In the last group an increase in the amount of plasma bicarbonate is the cause of the alkalæmia, and this may be an attempt to maintain the osmotic pressure of the plasma and to compensate for the excessive loss of sodium chloride which is taking place in the vomit or fæces. In some cases of diarrhœa and vomiting, however, an acidæmia may be present: this is due to some extent to water loss causing anhydræmia and defective renal circulation with consequent imperfect elimination of acid in the urine; in addition the impaired tissue circulation may lead to the formation of acid non-volatile products of incomplete combustion of carbohydrate and fat. Changes in hydrogen-ion concentration appear to act in the same way as carbon dioxide, i.e. mainly centrally and partly reflexly via the sino-aortic nerves [7].

Oxygen lack and breathing.—It is well to emphasize two preliminary points. Firstly, the inhalation of pure oxygen has no effect whatever on respiration. Secondly, cyanosis depends solely on the presence of excessive amounts of reduced hæmoglobin in the capillary blood and so is an index of the degree of oxygen want. It is entirely unrelated to the amount of carbon dioxide in the blood.

The effects of oxygen lack are complex and depend mainly on the degree of acuteness and severity of the deprivation.

(1) If an inert gas like nitrogen or nitrous oxide is breathed, the carbon dioxide is eliminated quite normally from the alveoli, but the oxygen which is present there is washed out as well. A certain degree of initial hyperpnœa occurs as a rule (though not invariably), but within 45 seconds, loss of consciousness develops suddenly, and practically without any warning, from oxygen lack to the brain. There are no reserve stores of oxygen in the tissues (unlike carbon dioxide); the only oxygen present there being the minute amounts which are present in solution. Soon after, breathing decreases and ceases from failure of the respiratory centre.

(2) If the oxygen lack is less severe, the effects produced depend on how rapidly it is induced.

If the subject breathes in and out of a large bag through a soda-lime chamber, the carbon dioxide evolved is removed by the soda-lime and does not therefore accumulate in the bag. The oxygen content gradually diminishes. The breathing is unaffected till the oxygen in the bag is reduced by about one-third, i.e. from 21 to 14%. In some sensitive subjects considerable increase in the breathing may now occur; but often the increase is too slight to be appreciable subjectively, though it can be shown to be taking place. It contrasts very strikingly with the intense hyperpnœa which results from even a small rise of carbon dioxide in the inspired air. Breathing is stimulated by oxygen lack, but to nothing like the extent to which it reacts to carbon dioxide excess. The effect of the increased breathing is again to raise slightly the oxygen content of the alveoli, and to wash out excessive amounts of carbon dioxide. As the blood becomes better oxygenated and its carbon dioxide content steadily falls, the breathing dies down again and never gets a chance to be maintained at a permanently high level. There is a tendency, too, for the breathing to wax and wane, i.e. be "periodic" in character. The effects may be summarized thus:—

Oxygen lack stimulates breathing, increases pulmonary ventilation, lowers alveolar carbon dioxide, lowers arterial carbon dioxide, produces alkalæmia, and so depresses breathing.

It is difficult to establish adequate compensation, as the stimulus of oxygen lack to breathing tends constantly to be counteracted by the alkalæmia. Cyanosis develops when the oxygen content of the inspired air falls to 10%, and this is accompanied by marked mental incapacity. Consciousness is lost a little later, from oxygen deprivation of the higher centres, without any air struggle.

(3) If the oxygen lack is of very gradual onset and ample time is available for all the compensatory activities of the body to be brought into play, the condition can be dealt with more effectively. This is well seen in gradual ascents to high altitudes. The essential point is that the kidney copes with the alkalæmia by excreting more alkali and making less ammonia. The blood reaction is in this way kept fairly normal and the ventilation is thus permitted to develop to, and remain at, the requisite high level. As a rule, the increase in breathing resulting from oxygen lack is chiefly in rate and to a less extent in depth.

Mode of action of oxygen lack [9].—As explained above, moderate degrees of oxygen lack stimulate breathing in the intact animal. But if the sinus nerves and vagi are severed, inhalation of the same mixture produces, as a rule, a rapidly developing paralysis of respiration. It can be concluded from this observation

that oxygen lack *directly depresses* the respiratory centre itself, but it acts on the sensory nerve-endings in the carotid sinus and the aortic arch, setting up excitatory impulses which *reflexly stimulate* breathing. As a rule, in the intact animal the reflex stimulation prevails, and increased ventilation results. It has already been pointed out how this hyperpnœa, by washing out carbon dioxide, sets up further complications. It is frequently stated that oxygen lack sensitizes the respiratory centre to the action of carbon dioxide, but this is incorrect. The "isolated" respiratory centre (sinus nerves and vagi cut) responds more vigorously to carbon dioxide excess alone than to carbon dioxide excess combined with oxygen lack.

Blood-pressure, cerebral circulation and breathing [6].—It has already been stated that a rise of blood-pressure in the intact resting animal reflexly depresses respiration, while a fall of blood-pressure reflexly stimulates breathing. The sensory zones which react to the blood-pressure changes are in the aortic arch and the carotid sinus. After section of the aortic and sinus nerves, blood-pressure alterations no longer affect breathing; this shows that changes in blood-pressure (so long as they do not lead to severe cerebral anæmia) have no direct effect on the respiratory centre. It is worth stressing that the respiratory centres, more than the other bulbar centres, rapidly lose their excitability if their oxygen supply is cut off or if their blood-supply is interfered with, as by raised intracranial pressure or by cerebral anæmia. Under such circumstances, if the blood-pressure is raised and the cerebral blood-flow is improved, rhythmic respiration may be restored.

Rôle of the vagi in respiration [3].—The vagal afferents which come from the laryngeal mucous membrane are concerned with the cough reflex and guard the respiratory passages against the entrance of foreign bodies. The afferent fibres which come from the substance of the lung undoubtedly regulate the rhythm of breathing.

As is well known, division of both vagi produces slowing and deepening of breathing. Paralysis of the laryngeal muscles is produced at the same time and causes obstruction of the air-way, which itself slows down breathing, but the slowing is obtained equally well when the vagi are divided in the tracheotomized animal, so that it is probably due essentially to the cutting off of afferent impulses from the lungs.

A good deal of light has been thrown on this subject by the studies of Adrian on the action currents (which are set up by nervous impulses) in single fibres of the vagus. It is found that a stream of impulses pass up during inspiration; the frequency of the impulses varies directly with the degree of distension of the lungs, and it dies down again during expiration. The impulses are probably set up by nerve-endings in the lung which are stimulated by stretch and which behave very similarly to the stretch receptors in muscle. If the lungs are artificially distended an impulse stream is set up (similar to that observed during normal inspiration) and it continues practically unaltered for as long as distension is kept up. *Forcible collapse* of the lungs, induced by sucking out the contained air, sets up another impulse stream which may arise from a distinct set of nerve-endings; probably, under normal conditions, they only come into operation very exceptionally.

The physiological effects of these vagal afferent impulses are shown by Head's experiments. If the lungs are forcibly distended, a prolonged expiratory effort is reflexly produced; if the lungs are collapsed a strong inspiratory effort is made. These results are not obtained after section of the vagi, proving that these nerves form the afferent limb of the reflex arc employed.

The conclusions which can be drawn from the above observations are as follows: The vagi signal the degree of expansion of the lungs; normally, when inspiration has proceeded to a certain degree, afferent impulses are set up in the vagi, which check inspiration, and consequently expiration takes place. It has been pointed out that the respiratory centre possesses inherent rhythmicity. When it is cut off from vagal afferents it discharges for a long time (giving a deep inspiration) and then passes into

a long rest-period during which it can recover to its original state. The presence of vagal afferents cuts short inspiration so that it becomes more shallow; as the period of activity is short, the time necessary for recovery is similarly abbreviated, and consequently the next inspiration follows more rapidly, so leading to an increased respiratory rate. As explained, afferent impulse streams are only set up very exceptionally during expiration.

Rôle of afferents from the cardiovascular area [7].—As has been pointed out the endings of the sinus and aortic nerves in the carotid sinus and aortic arch, respectively, are sensitive to changes in blood-pressure, the carbon dioxide tension, the hydrogen-ion concentration and the oxygen content of the blood. A fall of blood-pressure and a rise of carbon dioxide tension or of hydrogen-ion concentration, and oxygen lack reflexly (via these afferent nerves) stimulate the respiratory centre (see figure, p. 21). Conversely, a rise of blood-pressure and a fall of carbon dioxide tension or of hydrogen-ion concentration reflexly depress respiration. There is evidence [7] that the sensory nerve-endings which respond to alterations in the chemical state of the blood do not lie in the adventitia of the carotid sinus itself but in the closely related carotid body which contains numerous thin-walled vascular channels where the blood comes into intimate relationship with sensory nerve-endings.

Influence of respiratory centre on other bulbar centres.—Sinus arrhythmia (the variation in cardiac rate which occurs normally with respiration) has been shown to be due to an irradiation from the respiratory to the cardiac centre [10]. During inspiration the activity of the vagus centre is diminished and the heart quickens, while during expiration the discharge of the vagus centre is increased and the heart is slowed. It has further been shown [11] that the respiratory centre also irradiates directly to the vasomotor centre; during inspiration the discharge of the latter centre is increased, causing greater arteriolar constriction and a tendency for the blood-pressure to rise; the reverse process takes place during expiration.

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Respiratory Failure

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The term "respiratory failure" should have a wide interest for workers in all branches of medicine, since the commonest cause of death, if a final analysis be made, is "want of breath." The body has little or no power of storing oxygen, and if the apparatus responsible for obtaining this essential element fails there is always the possibility of a fatal result, while minor degrees of failure bring about varying states of disability. The term respiratory failure should, I suggest, be used in the same way as we use the terms cardiac failure and, more recently, renal failure. It is, perhaps, not so easy to study as the last-named which permits of a fairly simple chemical basis, and there is a further difficulty that the heart and circulation play an intimate part in the total respiratory processes of the body.

Respiratory failure in the new-born.—The conditions at this period of life are peculiar and in many ways the problem is simpler than in later years. At a discussion on the subject in Canada, recently, Dr. W. Easson Brown [1] said :—

"I also believe that this department of obstetrics should be looked after by those engaged in the speciality of anæsthesia. It seems to me that with their knowledge of intratracheal intubation and insufflation, and their constant use of various gases, they are eminently qualified to be of immeasurable assistance to the obstetrician in cases of failure of respiration in the new-born."

The following may be regarded as the principal causes of respiratory failure in the new-born, roughly in order of frequency :—

(1) Obstruction in the respiratory tract. (2) Delayed expansion of the lungs. (3) Narcosis. (4) Circulatory failure. (5) Intracranial damage. (6) Failure or delay in function of the respiratory centre.

The first two on this list are intimately related. Recent studies [2] have shown that expansion of the lungs after birth is by no means as rapid an affair as was originally thought. Far from opening up completely with the first breath, the alveoli in the normal infant probably take several days to expand properly, and considerable force is necessary to separate moist and adherent surfaces. It follows that even a small degree of obstruction may gravely interfere with this process and not nearly enough emphasis is laid upon the adequate clearing away of mucus and débris from the upper air passages. In the otherwise healthy infant the glottis probably protects the trachea and bronchi from invasion, but with intracranial damage leading to complete loss of muscular tone throughout the body, this protective mechanism may be lost. The old-fashioned metal mucus catheter is a difficult instrument to use and may be dangerous in unskilled hands. A rubber tube seems to me preferable from every aspect and can, if necessary, be passed into the trachea in order to clear obstruction in the lower parts of the respiratory tract. The same tube can be used to deliver gas mixtures under measured pressure, to aid in the expansion of the lungs. It is also obvious that methods of artificial respiration are useless unless the air-ways are clear. A combined suction and air delivery unit would seem to be a desirable piece of equipment at every maternity hospital, and I am hoping shortly to experiment with an apparatus of the type recently described by Mollison [3].

Narcosis as a cause of respiratory failure in the new-born is not uncommon after morphia has been given to the mother. Such babies are blue at birth and breathe feebly, with long pauses of apnœa. Cutaneous stimulation generally seems to make matters worse, but gas resuscitation with carbon-dioxide-oxygen mixtures is very satisfactory. The respiratory centres are probably depressed by the narcotic, so that a larger concentration than normal of carbon dioxide in the blood is necessary to stimulate them into activity. Shute and Davis [4], in a recent study on this subject, recommend pure carbon dioxide followed by pure oxygen administered with a mask. In skilled hands this is no doubt satisfactory, but I believe lower concentrations of carbon dioxide are desirable and that a mask is unnecessary. Five per cent. carbon dioxide in oxygen seems to produce good results and I see no particular reason for using higher concentrations as a routine. This is best administered through a nasal catheter. The effects of circulatory failure and of intracranial damage open up problems which would take us away from the main topic. The last cause on the list also raises very interesting points as regards the "unripe" condition of the centres in many infants, and has been well discussed by Peiper [5].

Certain interesting physiological principles can be followed in disorders of the respiration of the new-born. For example, it is often argued that it is absurd to administer carbon dioxide to a deeply cyanotic baby. It should be clearly realized that cyanosis has nothing directly to do with the amount of carbon dioxide in the blood. It depends entirely upon the absolute amount of reduced hæmoglobin present,

and when this reaches a certain threshold cyanosis appears. Cyanosis in the new-born is more easily produced because of the polycythæmia and raised percentage of hæmoglobin present at birth and may occur without either any serious degree of anoxæmia or of alteration in the carbon dioxide content of the blood. The depressed respiratory centre requires a higher percentage of carbon dioxide to stimulate it and the cyanotic baby often has a depressed centre from various causes. It has been claimed that animals placed for prolonged periods in an atmosphere with an increased carbon dioxide tension adjust their breathing to this new set of circumstances, and on being returned to a normal atmosphere show alarming failure of respiration often leading to death. The normal level of carbon dioxide is not sufficient to keep the control mechanism at work. Such a state of affairs is also to be seen in the new-born babe and also, possibly, after certain forms of anæsthesia when some degree of increase in the carbon dioxide tension of the inspired air has been present.

A second set of circumstances likewise common to the new-born and to the anæsthetized patient is that dependent upon the "Bohr phenomenon" [6]. Put very simply, this means that, within limits, oxygen is better liberated from the hæmoglobin of the blood, the higher the partial pressure of carbon dioxide. Now hyperventilation of the lung, whether brought about by artificial respiration, ether anæsthesia, or other causes, washes out carbon dioxide from the lungs and lowers the level of this gas in the blood. Thus artificial respiration of a cyanotic baby may sometimes remove the cyanosis, but by lowering the carbon dioxide tension in the alveoli, and thus in the blood, it interferes so grossly with the liberation of oxygen in the tissues that a serious and often fatal degree of anoxæmia may develop. This danger of "acapniæ" can be adequately prevented by the use of carbon dioxide mixture as the inhalant gas for resuscitation.

These foregoing remarks are not intended to be in any way a comprehensive account of respiratory troubles in the new-born. They are merely an attempt to indicate that certain general principles govern the mechanism of respiratory failure at all ages and in all circumstances. Whether a baby is "blue" or "white" is not really so important as a knowledge of the causation of the disorders of breathing.

Respiratory failure in general.—In attempting to classify the causes of respiratory failure in general terms, I must acknowledge my gratitude to Professor L. Brauer, of Hamburg, under whose supervision I was privileged to work some short time ago.[1] Some of the results of our work in his clinic have already been published [7], [8] and the following classification is largely based upon a review of the subject of respiratory failure by Brauer himself [9] :—

(1) Central failure. (2) Peripheral failure : (*a*) Mechanical (muscular, disease of lungs, etc.) ; (*b*) Interference with ventilation in respiratory passages ; (*c*) Interference with gaseous exchange between alveoli and blood ; (*d*) Circulatory disturbances in the lung, short circuits ; (*e*) Alteration in composition of inhaled gases.

This is, at the most, a very rough classification, but it will serve as a basis for a discussion of various points.

Respiratory failure of central origin is of great importance, but comparatively little is known of the exact way in which it is brought about. The *nutrition* of the centre is of importance and if the blood supply is deficient, depression of the centre will occur. This is seen in certain cases of heart failure in which, despite the absence of any gross change in the lungs, breathing is rapid and shallow. The oxygen content of the blood is normal (until the patient becomes moribund) and the usual explanation offered is that of stagnation at the respiratory centre (Fraser [10]). More severe degrees of heart failure when there is diminished oxygen in the blood, give rise to a curious periodical stimulation of the centre, leading to one type of Cheyne-Stokes

1 While holding a Rockefeller Medical Fellowship, 1930-31.

respiration. Interference with the nutrition of the centre may also occur with increased intracranial pressure and the resulting anæmia of the medulla causes depression of the respiratory centre. Cheyne-Stokes breathing again occurs in this form of disorder, probably owing to associated changes in the vasomotor centres. In the new-born, increased intracranial pressure due to œdema is one of the factors producing disturbances of respiration. The work of Peiper [5] suggests that the first effect may sometimes be the depression of parts only of the respiratory centre, leaving the "gasping centre," for example, intact, so that a completely new type of ineffective respiration results. The question of the possible influence of the cerebrum on the respiratory centre has recently been resuscitated, but the evidence is more suggestive than conclusive. The occurrence of Cheyne-Stokes breathing during sleep, for example, in certain ill patients has suggested that cerebral control can prevent this type of breathing. Probably simple depression of an already abnormally nourished centre will explain this, however. The irregular forms of respiration after encephalitis lethargica must also be taken into consideration. In one patient in our German series who had gross cerebral degeneration, periodic breathing, resembling Cheyne-Stokes in type, was present for long periods. It is also of interest that these cerebral types of what Knipping has called "pseudo-Cheyne-Stokes" breathing were not influenced by the inhalation of oxygen. (The effect of carbon dioxide was not investigated.)

Another important cause of central failure of respiration occurs when the centre is *poisoned*. Certain endogenous toxins may cause this, as in uræmia, in which irregularities of breathing may occur. The effect of sedative drugs, such as morphia and members of the barbiturate group, is of great importance. The centre may be so depressed that the accumulation of carbon dioxide consequent upon the shallow irregular breathing fails to exert its usual influence. Administration of oxygen alone to these patients, who are usually cyanotic, is of not much value even if means are taken to get it into the alveoli and thus to the blood. The use of carbon dioxide mixed with oxygen is much more effective, but even this may fail with a badly damaged centre. (Treatment is referred to later.)

Turning next to the *peripheral* causes of respiratory failure I would like to discuss particularly the possible faults in the ventilating power of the lungs. The pulmonary apparatus is essentially a bellows mechanism, concerned with taking air in and getting rid of the mixture produced in the alveoli. If the anatomical structure of the bellows is disordered, their efficiency is impaired, and yet such is the reserve-power of the lungs as regards pulmonary tissue, that gross anatomical changes may be associated with excellent ventilating efficiency, while normal structure may be accompanied by poor performance. Muscular failure, as when the diaphragm or intercostals become paralysed, produces an obviously serious state of affairs which used to be quite hopeless to treat until the Drinker apparatus was invented and cases of temporary paralysis could be tided over. Small premature babies, with feeble respiratory muscles, can also be kept alive in this way. Disease of the lung producing pure mechanical alteration in respiration is a somewhat complicated problem. One of the first effects is usually a reduction in the vital capacity. This is the total amount of air which can be expired after a maximum inspiration. With the addition of the "residual air" which cannot be expelled, it represents the maximum capacity of the pulmonary spaces. Obviously, a neoplasm—for example—growing and destroying a whole area of lung, must reduce the vital capacity, but the degree of lung damage is not proportional to the extent of reduction. Even a small area of active tuberculosis will often produce a striking reduction in the vital capacity, while on the other hand, I have seen a muscular young man with a spontaneous pneumothorax, whose vital capacity was actually above the normal for his age and body-build. Asthma is a disease which seems to produce a marked decrease in vital capacity, out of proportion to the amount of tissue damage in the

lungs, probably on account of the interference with the expiratory phase of the respiratory cycle. It would be possible to estimate the degree of respiratory failure in terms of vital capacity, but, as I have shown above, this would not be a sufficiently accurate guide taken alone, since mechanical failure can, to a certain extent, be compensated for by efficient ventilation.

During the past two years I have been especially studying this problem of ventilation [1] to see if an estimation of respiratory efficiency could be based upon this function. The amount of air breathed in at each breath is known as the tidal air, and this, multiplied by the rate per minute, gives what is known as the minute volume of respiration. This will vary in quantity, firstly according to the age and body build of the patient, and secondly according to the bodily requirements in the way of oxygen (and of carbon dioxide excretion). One way of estimating the ventilating power of the lungs is to take a standard quantity of oxygen, say 100 c.c., and work out how much air it is necessary to inspire in order that the body may obtain this quantity of oxygen. In other words, by means of a recording spirometer we can measure the minute volume and the oxygen consumed per minute and work out the volume breathed on a basis of 100 c.c. of oxygen. This index which has been called [7] the "ventilation equivalent for oxygen" is normally about 2·5 litres. It is lowered in the shallow breathing of morphia-poisoning and raised in many lung conditions, such as tuberculosis. But unfortunately this index does not take into account two other factors. One is that hyperventilation may be present which has nothing to do with oxygen requirements. Thus in diabetic coma, Knipping and I found the highest values in our series and this greatly raised ventilation equivalent had nothing to do with decreased respiratory efficiency. The second point is that the same minute volume can be obtained by rapid shallow breathing or by slow deep breathing, and these two extremes of respiratory movement are by no means equally efficient. This led me to a study of another aspect of the problem covered by the term "dead space."

The expired air issuing from the nose or mouth is a mixture of "lung" air from the alveoli and inspired or atmospheric air which has never reached the alveoli and has never been subjected to the true respiratory processes. This portion of the expired air is said to have come from the "dead space." It is obvious that there is a strict anatomical interpretation of this term for the upper air passages and the bronchi can take no part in the respiratory exchange between air and blood. But this is not the whole story, for in inefficient forms of respiration the amount of air in the lungs not entering into true pulmonary respiration is often greatly increased, and when we say that the "dead space" is increased in such cases we do not necessarily mean a strict anatomical increase. I have set out the evidence for this elsewhere in some detail [11]. For the purpose of this paper all that matters is that there is a normal optimal proportion of the "tidal air" which is "dead space." This is about 30%. In inefficient ventilation this percentage may rise to over 60. Put the other way round, it may be said that in the normal individual 70% of the air inspired at each breath actually takes part in the respiratory processes. This may be looked upon as the maximum degree of efficiency of the pulmonary bellows. It is decreased as the "dead space" increases. One interesting point regarding respiratory failure when estimated in this way, is that while at rest, with normal respiration, the pulmonary ventilating efficiency may be normal and remain at more or less the same level when the breathing is greatly increased by exercise, the same is not true where hyperventilation occurs as a voluntary forced effort. Then the efficiency drops considerably (i.e. the so-called "dead space" is increased). This unsatisfactory effect of forced hyperventilation should be borne in mind in association with the other phenomenon arising from this cause described above, in connexion with the dissociation of hæmoglobin.

1 Working with a part-time personal grant from the Medical Research Council.

To sum up this type of respiratory failure, it may be said that decreased pulmonary ventilation occurs when the patient is not breathing at the optimum level as regards tidal air and rate. This may be due to functional causes and is in that case presumably temporary. For example, the patient with spontaneous pneumothorax mentioned above, with the vital capacity above normal, had a "dead space" which was 65% of his tidal air, that is a pulmonary ventilating efficiency of 35%. On the other hand I have estimated the respiratory efficiency of a woman with asthma, whose vital capacity was considerably lowered, yet her "dead space" was only 35% of her tidal air, and the pulmonary ventilating efficiency thus almost normal. Some patients exhibit both a poor anatomical reserve, so to speak—as shown by a decreased vital capacity—and a poor pulmonary efficiency and here obviously respiratory failure cannot be expected to improve. A patient with a neoplasm of his lung had a vital capacity reduced by over 30%, and the "dead space" was 52% of his tidal air. I have not investigated the respiratory efficiency of patients under the influence of anæsthetics—owing to obvious practical difficulties—but I should expect to find them rather in the group with normal anatomical (i.e. mechanical) reserves, but diminished pulmonary ventilating efficiency as shown by an increased "dead space." In other words, the patient who is "breathing deeply" may actually be ventilating the alveoli less efficiently than when breathing quietly at rest.

The remaining causes of respiratory failure on my list may now be briefly dealt with. The part played by the alveolar epithelium in the all-important gaseous exchange between air and blood has been the subject of a certain amount of controversy into which I do not propose to enter. The balance of evidence is against any vital secretion of oxygen into the blood by the alveolar cells, but obviously disease of these cells may interfere considerably with the passage of oxygen into the blood so that, despite adequate ventilation of the pulmonary tissues, blood passes through the lung capillaries and into the general circulation with the oxygen content very slightly raised. If this condition of the lungs is at all widespread, fatal degrees of cyanosis and anoxæmia will occur. This was to be seen in the influenza epidemic of 1918, when patients were described as going black in the face before death. Irritant gases may damage the alveolar epithelium in the same way. Brauer has suggested the name "pneumonosis" for this condition, and in his list of causes he includes ether anæsthesia. It is a type of respiratory failure which is particularly amenable to relief, if not always to cure, by the use of oxygen.

If the lung disease is such that the circulation of the blood is impaired through the damaged area, a different set of circumstances is present. In extreme cases the circulation of the blood from the pulmonary arteries to the pulmonary veins is shut off completely, the nutrition of the lung being kept going by the bronchial arteries from the systemic circulation. Since there is no venous blood flowing through the damaged portion of lung, there is no unoxygenated blood flowing back to the left auricle, and thus, in the more extreme degrees of "lung damage" (e.g. completely collapsed lung with a pneumothorax) there may be little or no interference with respiratory efficiency. In one small series of experiments in Hamburg, I found that a patient's ventilating efficiency was better very soon after an artificial pneumothorax had been performed than it was before, when the only lesion was a small area of tuberculosis.

The final cause of respiratory failure in my list refers to the composition of the inspired air. There are many fascinating problems concerned with this, especially as regards the respiratory efficiency of airmen and others living and working at high altitudes. The increase in ventilation necessary for the body to secure sufficient oxygen out of the rarefied atmosphere causes an excessive washing out of carbon dioxide with very important effects, some of which have already been mentioned. It is a condition which, I suppose, is also a possibility under certain conditions of faulty anæsthesia and is fortunately readily amenable to the use of oxygen.

Treatment.—The treatment of respiratory failure can only be carried out when a satisfactory analysis of the cause and type of this condition has been made. Central causes of failure present the more serious problems, as it is difficult to stimulate a centre which has been depressed by drugs, or has been exposed to higher concentrations than normal of carbon dioxide. Frankly, I know of no drug which can be regarded as a satisfactory stimulant to the respiratory centre. Atropine is supposed to counteract the effect of morphia, but I would rather use it before, than after, the centre had been paralysed. Drugs of the lobeline type certainly appear to have a stimulant effect somewhere in the nervous mechanism of respiration, but the pharmacologists rightly warn us of the after-depressant effect of members of the nicotine series. Carbon dioxide in oxygen seems to be the most efficient stimulus we possess, provided that the circulation has not failed, for the gas must be carried to the centre and respiration must be efficient enough to secure that the gas gets into the alveoli. It follows that the treatment of respiratory failure is intimately dependent upon the treatment of circulatory failure. The second necessity raises the problem of artificial respiration. It is obvious from what has already been said, that attempts at forced ventilation of the lungs which wash carbon dioxide out may fail in their purpose for this reason. I consider trachæal intubation (or the use of a mask) and rhythmical pumping of carbon dioxide and oxygen mixtures as preferable to artificial respiration when skilled aid and the necessary apparatus are available. Such ideal conditions are, of course, rarely present, and then artificial respiration properly carried out is the most important method available. It is a matter for regret that so few students and medical practitioners are really efficient at this procedure. The percentage of carbon dioxide to be employed for resuscitation is debatable. For babies I believe 5% is safe and satisfactory. For general use 7% is probably best. Pure oxygen is a valuable life-saver in certain forms of respiratory failure, especially when the pulmonary epithelium is diseased and in all cases in which anoxæmia is the primary and fundamental cause of the condition.

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Dr. Ernest Fletcher said that one of the first signs of pulmonary disease was an inability to expire the reserve air in the lungs, and this might be due either to plugs of mucus in the bronchi, producing a reflex tone in the muscles of respiration, so that the chest was held in a position of semi-inspiration, or to mechanical factors, such as the calcification of costal cartilages, which would not allow the thoracic cage to assume the position of complete expiration. The classical advanced example of this early condition was hypertrophic emphysema.

In 100 apparently normal cases which he had investigated, inspiration represented 30% of the respiratory cycle, and this figure was not altered in early pulmonary disease because, although the whole cycle of respiration was raised towards the side of inspiration, it was still balanced.

The injection of lipiodol into the lungs was rarely followed by complete respiratory failure, and emphysema—not collapse—was found; it was difficult to understand the part played by the blood gases and the respiratory centre in these cases.

Dr. R. J. Minnitt exhibited the following table compiled from his own observations, depicting the fall of carbon dioxide in the venous blood during anæsthesia :—

COMPARISON OF CO_2 FALL DURING DIFFERENT TYPES OF ANÆSTHESIA.

Case No.	Operation	Method	Amount of ether used	Time of anæsthesia	CO_2 vols. per cent. from —
117	Gastro-enterostomy	Open ether	20 oz.	60 mins.	56 to 51
119	Subtotal hysterectomy	Gas/oxy/ether endotracheal	1 ,,	60 ,,	52 to 46
120	Goitre removal	,,	2 ,,	55 ,,	58 to 53
121	Gastro-enterostomy	,,	3½ ,,	60 ,,	60 to 54
118	Cholecystectomy	Gas/oxy/ether face mask	3 ,,	60 ,,	58 to 54
122	Appendicectomy	,,	4 ,,	45 ,,	56 to 47
123	Appendicectomy	Gas and oxygen face mask	No ether	25 ,,	53 to 45
124	Rectal examination	,,	No ether	20 ,,	60 to 58
*129	Appendicectomy	Gas/oxy/ether face mask	2 oz.	25 ,,	52 to 51

*Case 129 had CO_2 administered from "Sparklet" direct through face mask at conclusion of the operation.

NOTE.—Fairly constant fall of CO_2 irrespective of method used, with the exception of case in which pure CO_2 was administered.

INFERENCE.—Indication for re-breathing and use of CO_2.